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Essentials of Hand Surgery

Edited by Alexandro Aguilera Salgado

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Notice

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Meet the editor

Dr. Alexandro Aguilera is a plastic and reconstructive surgeon and a faculty member at the Department of Plastic and Reconstructive Surgery of the National Institute of Pediatrics in Mexico City, Mexico. He is a board-certified plastic surgeon from the Mexican Board of Plastic, Aesthetic, and Reconstructive Surgery. He won a fellowship in Hand Surgery and in Facial Palsy and Peripheral Nerve from the National Autonomous University of Mexico. He is a part of the Educational Committee of the Mexican Association of Plastic, Aesthetic, and Reconstructive Surgeons. He participated in numerous manuscripts, poster presentations, and books and contributed to several national and international plastic and reconstructive surgery meetings.

Contents

Preface

Understanding the essentials of hand surgery as well as the complexity of the mechanisms of injury and the specific reconstructive goals is mandatory for every hand specialist involved in this passionate area. The objective of this book is to update hand specialists on the diagnosis and treatment of some of the most common pathologies affecting the hand and also to provide new insights and recent advances in this field. We have gathered a group of clinicians and scientists with a large experience in hand surgery. This book is divided into two sections. Section 1 is called "Basic Principles," and it includes two chapters. In Chapter 1, we discuss the importance of hand surgery and the diagnostic methods, such as how to plan our operation, special considerations during surgery, and post-operative care. In Chapter 2, Dr. Harsha and collaborators reviewed the techniques for regional anesthesia in hand surgery. Section 2 is called "Reconstructive Hand Surgery," and it includes five chapters. In Chapter 1, Dr. Justin and collaborators reviewed the anatomy of flexor tendons, the diagnostic process of flexor tendon injuries, and their treatments. In Chapter 2, Dr. Benavides and collaborators reviewed the diagnosis and current treatment of carpal tunnel syndrome. In Chapter 3, Dr. Jeremiah and collaborators reviewed the pathophysiology in Dupuytren's disease as well as the different treatment options we have and future trends in this area. Finally, in Chapters 4 and 5, Dr. Roohi and collaborators reviewed crush injuries of the hand, establishing interesting options for the treatment of these injuries with difficult cases being solved excellently.

I dedicate this work to my family—mom, dad, sister, and my fiancée. They are the greatest pillars I have in my life and the ones that have made me what I am now, a responsible, honest, thirsty-for-knowledge man, who never gives up on anything, thank you for your support and for being a source of inspiration. I would also like to thank my mentors and colleagues for pushing me very hard day by day to achieve my goals. Last but not least, I would like to thank my patients because without them none of this would have been possible. Thank you all very much.

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Section 1

Basic Principles

Chapter 1

Introductory Chapter: The Art of Hand Surgery

Alexandro Aguilera

Additional information is available at the end of the chapter

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1. Introduction

Hand and upper limb surgery is one of the most difficult areas in plastic and orthopedic surgery. It demands thorough knowledge of normal hand anatomy and great expertise in adequately diagnosing and treating the multiple pathologies that can affect the hand, followed by the selection of the best individualized postsurgical hand therapy, in order to lead our patients into the reestablishment of their hand's function.

A strong functional hand with normal range of movements is the main characteristic among human beings. With our hands we can perform our normal daily activities, interact with other people, perform our jobs, play musical instruments, and participate in most of our activities. Our hands and fingertips are the doors to the world, being so adaptable, that they can even make a blind man read or a deaf-mute speak. This is why we must approach each of our cases with artistry, trying to recover maximum mobility, sensibility, stability, and strength possible, getting rid of all of the pain the patient may have, in order to regain its function and unique esthetics [1].

It is mandatory for a hand surgeon to have some special characteristics. He or she must have sound surgical skills, a natural sense of artistry, must be an expert in hand anatomy, must be imaginative and creative in order to perform and design new techniques, should be delicate enough with the different hand tissues, should pay careful attention to the most minimum detail, and have the maximum technical precision in order to be successful. With the invention of the microscope and the refinement in surgical instruments we can now perform surgeries we have never even dreamt, be less aggressive, and achieve better and amazing results with each surgery.

2. Performing the right surgery

As in every aspect of medicine, the key to success in hand surgery is to make an accurate diagnosis based on a complete history and physical examination of our patient. Once you have an exact diagnosis you will know exactly what to operate and most importantly when to perform that operation. We, as surgeons, have that rush to try to solve everything as soon as it is presented to us. We know we can solve things, but we also have to be very patient to know the perfect timing and when to do it in order for our surgeries to be successful and to achieve the best results possible.

3. Diagnostic methods

Besides our own eyes and hands, there are some other special diagnostic tests we can use in order to finally determine the exact problem. One of the most common and routinely performed tests are simple plain hand X-rays. As we know, the hand is a complex structure where many anatomic elements interact with each other. It is not just skin and subcutaneous tissue; we have bones, ligaments, tendons, nerves, arteries, veins, muscles, and joints that when acting together the hand will achieve a normal and complete function. There are some other special tests that can be asked in some special situations, like CT scans, MRI, electromyography, nerve conduction studies, Doppler ultrasound, and so on. As we have said the diagnosis must be made by us, not be expected to pop out spontaneously by some random and unnecessary studies.

4. Planning the operation

Once you have an accurate diagnosis it is plan for the next step, planning the operation. It is not enough for a hand surgeon to have just a plan for the surgery. You must have a plan B and a plan C and just as many as you need in order to act if something goes wrong. It is just not a matter of performing the surgery; it is a matter of knowing what to do and how to do it. From the incision planning to the closure of the skin, each step must be taken into consideration. Even though you are an experienced hand surgeon, our advice is to always plan your surgery the night before you perform it when it is possible. There is no small surgery; even the ones considered “easy” could turn into difficult challenges for the surgical team [2].

5. Special considerations during surgery

As you plan your surgery, there are some important aspects you may want to take into consideration that will surely affect the course of your performance. First, the incisions you plan are important, because you need the best exposure you can get with the incision you

Figure 1. Use of microscope, microsurgical instruments, hand table and controlled ischemia of the hand during surgery.

plan. Other important questions are as follows: What type of anesthesia will you use? Will you use local anesthesia? What nerves will you anesthetize? What is the type and duration of ischemia of the hand? Will you have pauses between periods of ischemia? At what pressure will the tourniquet be inflated? Is a microscope needed for the procedure? Is there any special equipment or instruments you may need? Do you need microsurgical instruments? Do you need equipment for bony fixation? Do you have your surgical loupes or some kind of magnification? Will you need X-rays to be taken after bony fixation? Is every suture you are going to need ready? What type of cast or immobilization will you use after the procedure? These questions ideally must be answered during your planning as if it was a checklist so it will be easier for you to make sure everything is ready before the surgery takes place (**Figure 1**).

6. Post-operatory care

Once you have completed your surgery, you have to decide if your patient may need a special rehabilitation program so he or she can get the best functional result possible. For some procedures, it would just be enough with some exercises the patient can perform at home. For some others, you may need special casts or immobilization, followed by specialized hand therapy. It is important to discuss these questions with the patient before the procedure takes place. As I usually say to my patients, the results depend 50% in the surgery itself, and the other 50% will depend on the patient itself and on the hand therapist. This specialized work is not just a one-day job. It is mandatory that the hand surgeon reevaluates his patient as many times as needed until his or her function has been completely reestablished.

7. Conclusions

The aim of this chapter is to provide the reader with a comprehensive and state-of-the-art overview of the normal hand and upper extremity, its physical examination, how to make an accurate diagnosis of the diverse situations that can affect it, and to discuss the most important evidence-based methods of treatment available worldwide today.

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Chapter 2

Regional Anesthesia for Hand Surgeries

Harsha Shanthanna and Manikandan Rajarathinam

Additional information is available at the end of the chapter

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Abstract

Anesthesia for hand surgeries is one of the domains of regional anesthesia, where it plays the role, not only as a viable alternative to GA but also as an adjunct to it. The efficacy and inherent advantages of regional anesthesia techniques are widely made use of by the hand and upper limb surgical centers across the world. There are a variety of established regional techniques ranging from major plexus blocks to local infiltration techniques and intra venous regional anesthesia for hand surgeries. The peripheral nerve blocks are recently being performed with ultrasound guidance which is undoubtedly the greatest influence till date in the practice of modern regional anesthesia. There is also a recent trend in the performance of certain minor hand surgeries (e.g., carpal tunnel release) under infiltrative techniques in office-like settings for rapid turnover. This chapter discusses concisely the selection and execution of such techniques in day-to-day practice.

Keywords: regional anesthesia, hand surgery, brachial plexus blocks

1. Introduction

Hand surgeries are amenable to regional anesthesia techniques which are often performed by the anesthesiologists. The ability to perform peripheral nerve blocks is a desirable skill to acquire by the anesthesiologists who provide anesthetics for upper limb surgeries. The advent of ultrasound guidance for these peripheral nerve and plexus blocks has revolutionized the practice of regional anesthesia in the past two decades. The enhanced accuracy and safety with ultrasound guidance offer higher success rate and reduced block latency, thereby improving the operator confidence.

2. Advantages and limitations of regional anesthesia

The practical advantages of regional anesthesia are faster postoperative recovery, better postoperative analgesia, avoidance of opioid-related side effects, and prevention of GA-related complications in patients with cardiovascular and respiratory comorbidities, e.g., sleep apnea, chronic obstructive pulmonary disease (COPD), coronary artery disease (CAD), etc. The perioperative patient satisfaction and hospital efficiency are readily perceptible with the use of these regional anesthesia techniques albeit the sparsity of data on long-term outcome following surgery. On the other hand, the availability of sophisticated equipment, specialized operator training, requirement of a dedicated block room, and protected block execution time are some of the limitations with routine practice of regional anesthetic techniques. Though the use of ultrasound as a nerve localization tool has shortened the learning curve and boosted operator comfort level with peripheral nerve blocks, it does not the eliminate the procedure-related complications.

3. Techniques of regional anesthesia for hand surgeries

The understanding of the structure of the brachial plexus and the innervation of the upper extremity is paramount to choose the right approach of neural blockade to achieve successful surgical anesthesia or analgesia. Of the four major approaches to brachial plexus block, the axillary approach, infraclavicular approach, and supraclavicular approach are commonly used for anesthesia and analgesia for hand surgery, whereas the interscalene approach is mainly used for shoulder and upper arm surgery. The axillary block is popular with hand surgeries as it provides sufficient distal extremity blockade and is relatively simple to perform and devoid of serious complications. The supraclavicular and infraclavicular approaches provide better tourniquet tolerance and wider surgical anesthesia of the entire upper extremity by virtue of the proximal neural blockade. A recent systematic review concluded that the axillary, supraclavicular, and infraclavicular approaches had similar success rates when performed with ultrasound guidance and there was no difference in successful blockade irrespective of number of injections [1]. Further distal approaches like mid-humeral block and individual peripheral nerve blocks may offer sensory blockade and analgesia but may not cover the tourniquet discomfort. However, application of forearm tourniquet for hand and wrist surgeries may be more comfortable for patients receiving regional anesthesia [2, 3]. This will obviate the need for proximal brachial plexus blocks given primarily for tourniquet discomfort and will avoid disproportionately extensive and often prolonged sensory and motor blockade beyond that required for incisional analgesia.

3.1. Techniques of brachial plexus block

3.1.1. Axillary brachial plexus block

Axillary block is an excellent anesthetic option for elbow, forearm, and hand surgeries either as a sole anesthetic technique with or without sedation or as a supplement to GA. Axillary block targets the four major nerves of the upper limb (the radial nerve, ulnar nerve, median nerve, and musculocutaneous nerve) which are arranged around the axillary artery as a neurovascular bundle that is easily accessed through the axilla. The technique is relatively simpler using the axillary artery as the landmark around which the major nerves of the upper

limb can be localized using nerve stimulation or ultrasound. It is also devoid of complications like pneumothorax and hemi diaphragmatic paresis. The block performance time with axillary block is relatively longer than with the supraclavicular and infraclavicular blocks due to the higher needling time [4, 5]. Also multiple injection technique has higher success rate than single or double injection technique when a non-ultrasound-guided technique was used [6].

3.1.1.1. Technique

The patients' arm is abducted with elbow flexed and supported in this position to expose the axilla (**Figure 1a**). A high-frequency linear array transducer is used to obtain a cross-sectional view of the axillary artery just distal to the anterior axillary fold (**Figure 1b**). At this point the radial nerve is still in close proximity to the axillary artery above the conjoint tendon formed by the teres major and latissimus dorsi (**Figure 1c**). Distal to this level, the radial nerve exits posteriorly through the triangular space and is difficult to visualize(**Figure 1d** and **e**). The median nerve is often lateral or superior to the axillary artery, and the ulnar nerve lies medial or posterior to the artery. The musculocutaneous nerve usually exits brachial plexus earlier and is found lateral to the artery between the heads of coracobrachialis muscle or between biceps and coracobrachialis. Variations in the relations of these nerves can be very commonly seen, but the nerves can be identified by tracing their course distally [7].

After obtaining the image as shown in **Figure 1c**, the block needle can be inserted in plane with or without the nerve stimulation. The musculocutaneous nerve can be blocked first as it is located lateral to the artery and often encountered first while entering in plane. The nerves need to be surrounded by local anesthetic for the block to take effect satisfactorily. The radial, medial, and ulnar nerves can be subsequently blocked by redirecting the needle. The block can be performed with one needle entry occasionally requiring a second pass to target one of the nerves.

3.1.2. Infraclavicular block

Infraclavicular block is a brachial plexus block performed at the levels of the three cords of the brachial plexus which are arranged around the subclavian artery. The cords are named lateral, medial, and posterior cords based on their fairly consistent relation to the axillary artery (**Figure 2e**) [8]. With the exception of a few nerves that depart the brachial plexus at the level of roots and trunks, the cords comprise of most of the sensory and motor innervation of the upper extremity. Hence, this block is an excellent anesthetic technique for surgeries of the distal arm, forearm, elbow, and hand. This block is relatively easier to perform with the ultrasound as the neurovascular bundle is deeper making it difficult to perform with nerve stimulation technique. Though the risk of hemi diaphragmatic paralysis is very rare, the risk of pneumothorax is still present if the needle is directed blindly medially toward the pleura.

3.1.2.1. The technique

For this block the patients' arm needs not be abducted, but abduction may help with visualization of the neurovascular bundle by stretching the overlying muscles and also potentially displacing the neurovascular bundle away from the chest wall. Though a high-frequency linear array transducer often should suffice for this block, in obese or muscular patients, a low frequency curvilinear transducer may be required. The probe is placed in a parasagittal plane

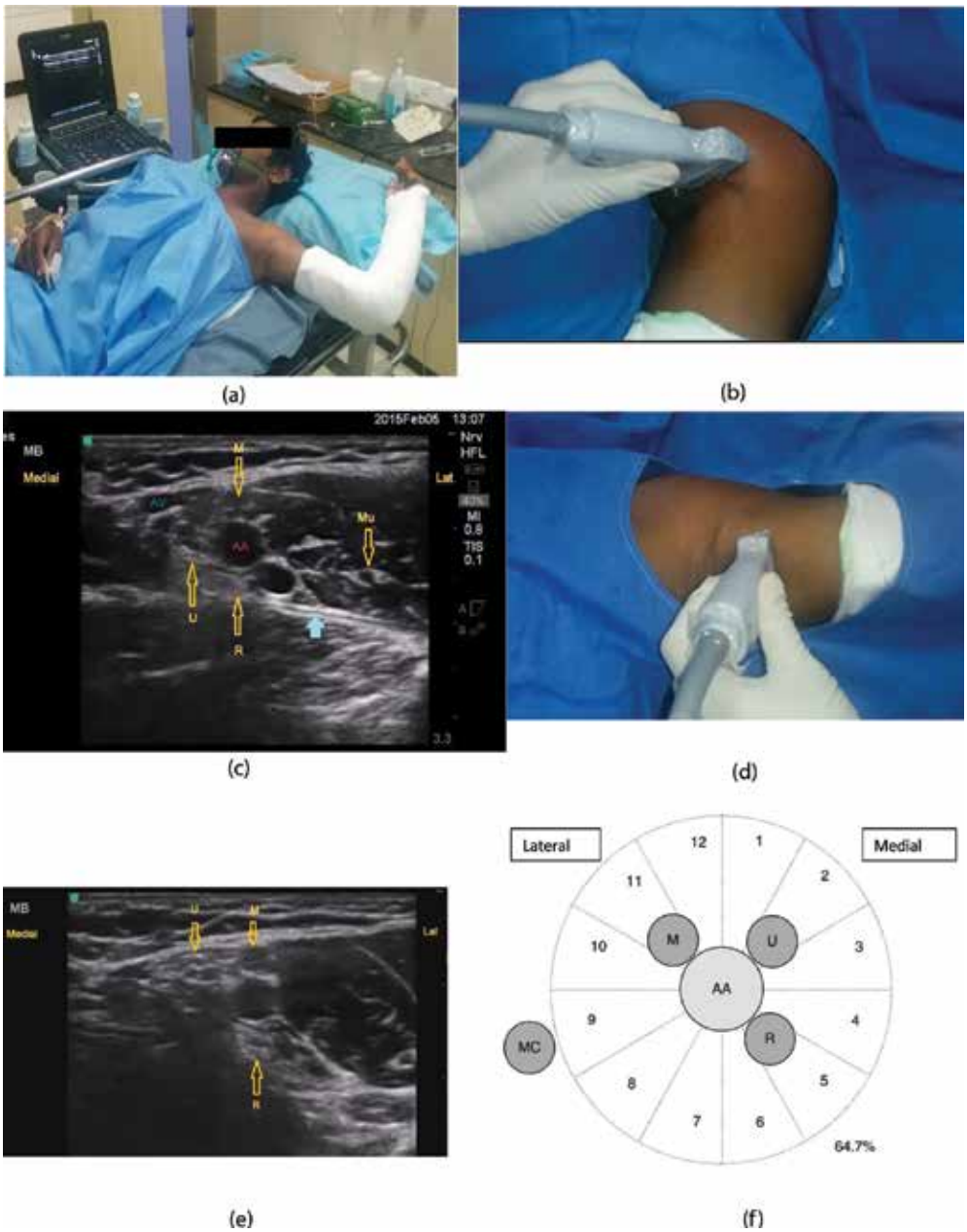

Figure 1. (a) Position of the upper limb with abduction of shoulder and elbow flexed to expose the axilla. (b, c) Linear probe placement close to the anterior axillary fold and the image obtained with the probe placed transversely across the neurovascular bundle higher up in the axilla where the conjoint tendon (solid arrow) is seen. The radial nerve is usually contained between the conjoint tendon and the axillary artery. Distal to this level, the radial nerve departs posteriorly and is difficult to visualize. AA, axillary artery; AV, axillary vein. (d, e) The image obtained with the probe transversely across the neurovascular bundle below the level of the conjoint tendon. The axillary vessels become brachial vessels below the level of the teres major. Note the ulnar, median, and radial nerves marked in the pictures with arrows. The radial nerve accompanies the profunda brachii artery and exits to the posterior compartment of the arm. BV, brachial vein; BA, brachial artery; M, median nerve; U, ulnar nerve; R, radial nerve. (f) The most common arrangement of the major four nerves around the axillary artery. Frequency of this pattern is 64.7%. Adapted from [7].

(**Figure 2a**) just medial to the deltopectoral groove to visualize the axillary artery in cross section and the circumferentially placed cords which are seen as hyperechoic structures (**Figure 2b**). After obtaining an optimal image, an 8 to 10 cm block needle is inserted in an inplane technique (**Figure 2c** and **d**) in a cephalocaudal direction aiming to place the needle tip posterior to the artery close to the posterior cord. A volume of 30 ml of local anesthetic can be injected to create a U-shaped local anesthetic spread around the axillary artery.

3.1.3. Supraclavicular block

Supraclavicular block is a brachial plexus block performed at the midpoint of the clavicle posterolateral to the subclavian artery where the distal trunks and proximal divisions of the brachial plexus are compactly arranged abutting the artery (**Figure 3a** and **b**). Due to this compact arrangement of neural tissue, the local anesthetic block latency is short and leads to anesthesia of the entire arm below the shoulder rapidly. This is an ideal technique for a sole regional anesthetic techniques for most surgical procedures of the entire upper extremity below the level of the shoulder. Due to the proximity of the pleura, there is a risk of pneumothorax with this block especially when performed with blind techniques. Recently, ultrasound guidance has shown to reduce the incidence of this complication due to real-time visualization of the structures while performing the block. There is also the possibility of ipsilateral phrenic nerve block with this block which might be symptomatic in some patients.

3.2. Individual nerve blocks

Radial nerve can be visualized easily in the distal arm in the anterolateral part just above the elbow (**Figure 4a** and **b**). It emerges from the spiral groove of the humerus and appears anteriorly between the brachioradialis and brachialis muscles. The nerve can be blocked here with 3 to 5 ml of local anesthetic to block its terminal branches of which the anterior branch supplies cutaneous innervation to the dorsal skin of the hand and lateral three digits. The median nerve accompanies the brachial artery throughout the arm, lending itself to easy identification with ultrasound scanning distally from the axilla. The median nerve can be blocked in the upper forearm where it lies between the flexor digitorum superficialis and flexor digitorum profundus in the center of the forearm (**Figure 5a** and **b**). The median nerve provides cutaneous innervation to ventral aspect of the lateral hand and fingers excluding the medial half of the ring finger and the entire little finger. The ulnar nerve can be visualized with ultrasound from the distal part of the forearm lying medial to the ulnar artery. It can be traced proximally and can be blocked at the upper forearm where it moves away from the artery, thereby avoiding injury to the vessel (**Figure 6a** and **b**). Prior to the use of ultrasound guidance, a landmark-guided technique at the olecranon fossa was described. However, the potential of nerve injury is high in this location due to proximity to bone and being enclosed in a fibro-osseous tunnel. The ulnar nerve innervates the skin of the hypothenar eminence and the ventral aspect of the medial one and half fingers.

3.3. Alternate techniques

Intravenous regional anesthesia or Bier block is an alternate technique ideal for short surgeries in the hand and forearm. A regional block is achieved by injecting the local anesthetic

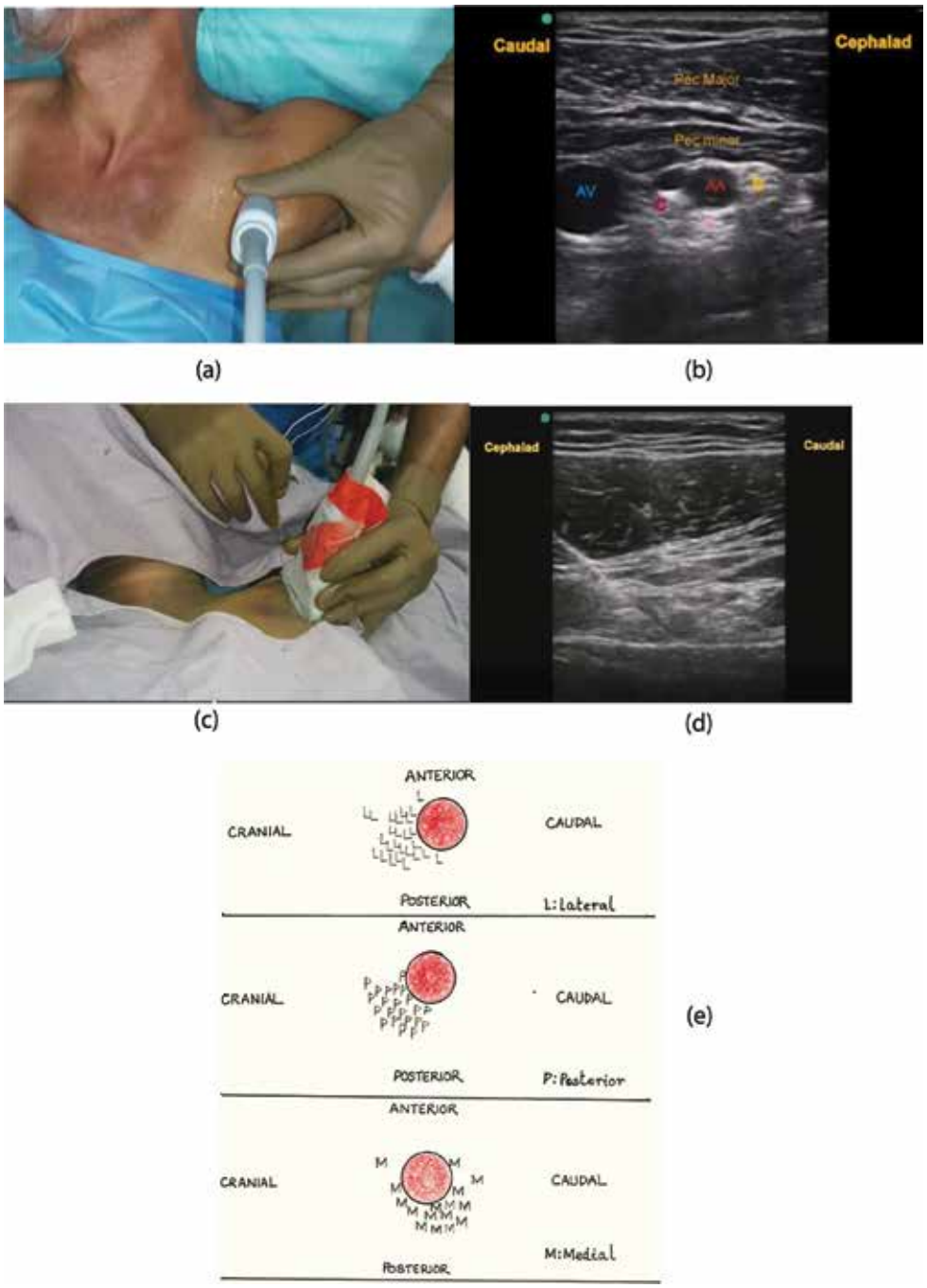

Figure 2. (a, b) Probe placement for infraclavicular block in the parasagittal plane in the deltopectoral groove. The axillary vessels are seen in cross section with the cords circumferentially arranged around the axillary artery. AV, axillary vein; AA, axillary artery; C, cords. (c, d) Needle inserted in plane in a cephalocaudal direction with the needle tip placed cephalo-posterior to the axillary artery. (e) The common arrangement of the cords around the axillary artery, adapted from [8].

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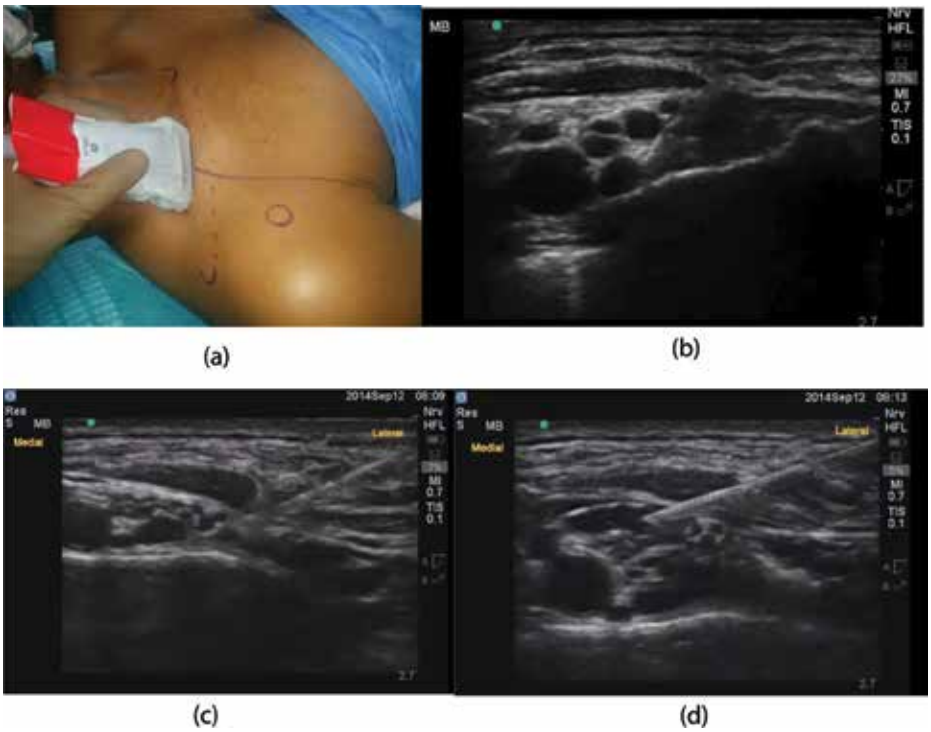

Figure 3. (a, b) Probe position for supraclavicular block and the resulting image showing the compact arrangement of divisions of the brachial plexus block abutting the subclavian artery and overlying the first rib. Note the pleura lying underneath the subclavian artery. (c, d) Performance of the supraclavicular block. The block needle is inserted inplane and directed between the first rib and the lower part of the plexus. The local anesthetic is also injected from above the plexus after redirecting the needle.

through the veins in the vicinity of the incisional site after application of an arm tourniquet and containing the local anesthetic distal to the tourniquet. The block is technically easy to perform, and the onset of the block is rapidly occurring within a few minutes.

3.3.1. Technique of IVRA

An additional temporary IV access is secured in the limb to be operated usually in the hand or wrist. Following this, exsanguination of the veins is done by elevating the arm and application of Esmarch bandage. After thoroughly exsanguinating the limb, a double-cuffed tourniquet with a proximal and distal cuff is wrapped around the arm. The distal tourniquet is first inflated followed by inflation of the proximal cuff. The distal cuff is then deflated leaving the proximal cuff inflated followed by slow local anesthetic injection in to the IV cannula. The block takes effect in a few minutes and usually lasts for an hour. If the patient complaints of tourniquet discomfort, the distal cuff is inflated, and the proximal cuff is subsequently deflated. The tourniquet needs to remain inflated for at least 30 minutes till the local anesthetic fixes to the tissues even if the procedure is completed before this time. Due to the risk of systemic local anesthetic toxicity, lidocaine is the agent of choice due to its relatively low cardiac toxicity. Application of

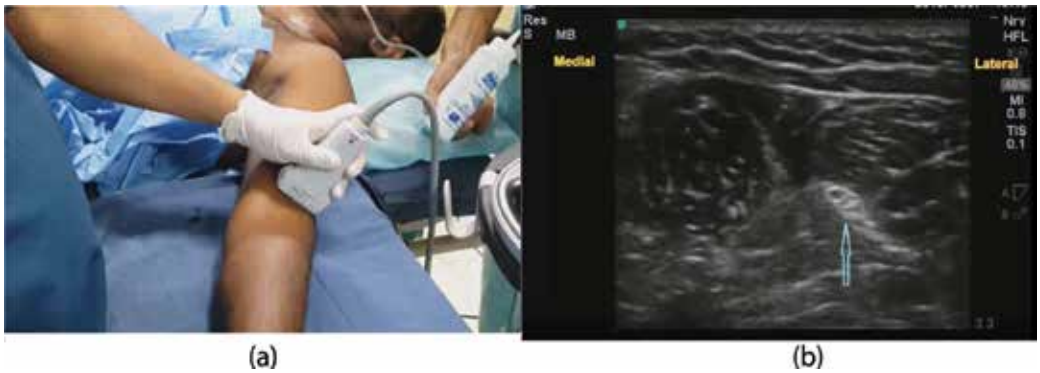

Figure 4. (a, b) Probe placement for visualization of radial nerve in the anterolateral surface of arm. The radial nerve is seen under the brachioradialis muscle.

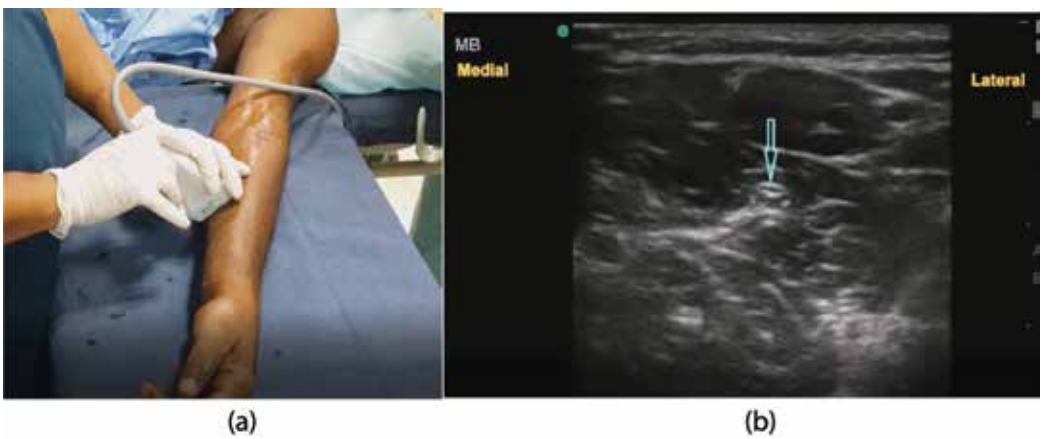

Figure 5. (a, b) Probe placement for visualization of the median nerve in the center of the anterior aspect of forearm. The median nerve is seen between the flexor digitorum superficialis and flexor digitorum profundus.

forearm tourniquet for hand or finger surgeries may be more comfortable for the patients and also can potentially reduce the amount of local anesthetic intravenously in IVRA [9].

3.3.2. Local infiltration techniques

As an alternate to the regional anesthetic techniques performed by anesthesiologists, some of the procedures can be performed by local anesthetic infiltration, wrist or digital nerve blocks by the operating surgeons.

4. Recent trends in hand surgeries

There is a recently increasing trend to perform some of the hand surgeries like carpal tunnel release, Dupuytren's release, trigger finger release, tendon repair, etc., to be performed under local anesthesia with no sedation/anesthesia and no tourniquet [10]. This improves hospital efficiency and

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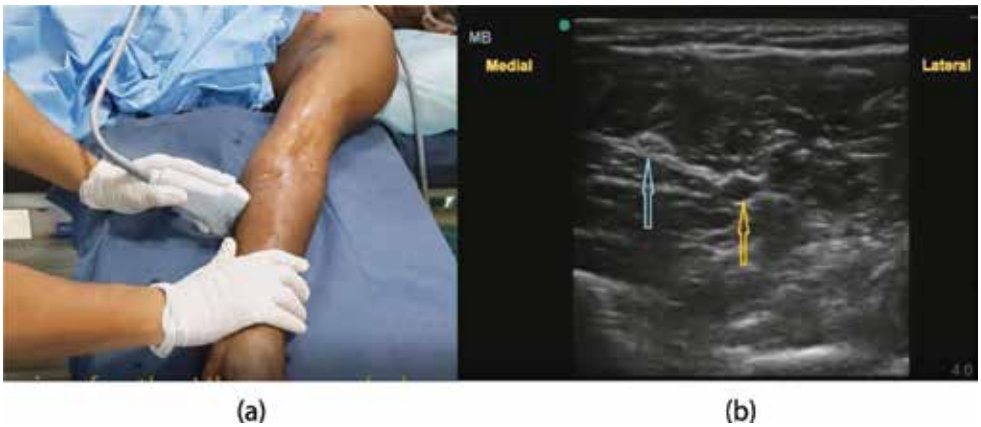

Figure 6. (a, b) Probe placement for visualization of the ulnar nerve in the upper medial surface of the forearm. The ulnar nerve will been seen accompanied by the ulnar artery laterally distal to the mid-forearm. The block is ideally performed when both structures are away from each other in the upper aspect of the forearm. The hyperechoic structure medially is the ulnar nerve, and the hypoechoic structure laterally is the ulnar artery which is approaching the nerve when traced distally.

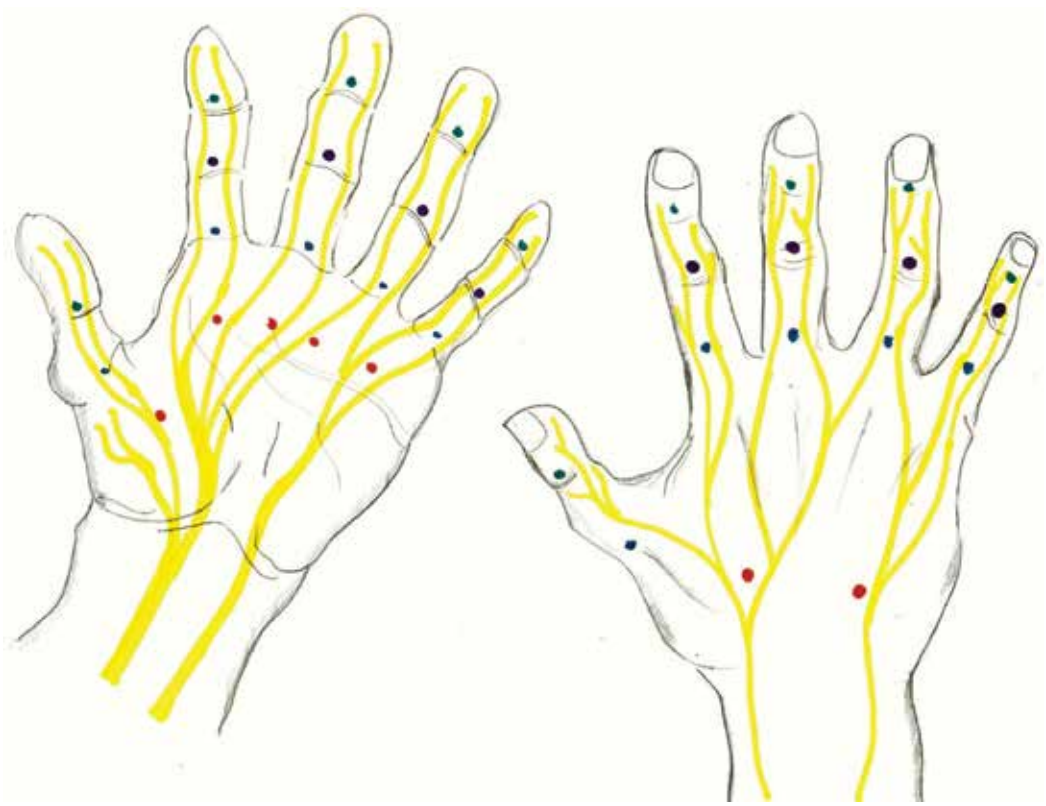

Figure 7. Illustration of volar and dorsal sites of injection of local anesthesia for finger and hand surgery. For only a sensory block of the finger, 2 ml is injected into the BLUE dot (third dot from the top) and is called the SIMPLE (single subcutaneous injection in the middle of the proximal phalanx with lidocaine and epinephrine) block. For local anesthesia and hemostasis of palmar finger surgery, 1% lidocaine with epinephrine 1: 100,000 is injected into the midline subcutaneous fat between the digital nerves at each area designated by a dot. From distal to proximal, the volume injected is 1 ml (first dot from the top), 2 ml (middle dots), and 5 ml at the red dots. Adapted from [11].

cost of surgery, reduces preoperative preparation/consultations, and helps fast tracking of these surgeries by performing in minor operating rooms. This also benefits certain procedures like tenolysis and tendon repairs allowing for monitoring of the finger range of movement intraoperatively. The technique typically involves subcutaneous injection of 1% lidocaine with 1:100000 epinephrine on the palmar and dorsal aspects of the finger (**Figure 7**) [11]. This type of wide-awake hand and wrist surgery is now popularly called (wide-awake local anesthesia and no tourniquet (WALANT) surgery [12]. The previous concerns of injecting epinephrine for hemostasis during hand and finger surgery for the fear of ischemic injury have been refuted recently by many investigators.

5. Conclusion

A multitude of approaches of local anesthetic blockade throughout the structure of brachial plexus and its branches are currently practiced to provide regional anesthesia and analgesia of distal upper extremity. The use of ultrasonography has not only revolutionized the conventional regional anesthesia techniques but also improved the access to the peripheral nerves at distal levels in the course of these neural structures. The major brachial plexus approaches like supraclavicular, infraclavicular, and axillary blocks have been shown to have equal success rates. Hence, the choice of the technique may be decided based on the experience of the practitioner, extent of the surgery, tourniquet use in the arm or forearm, and consideration of avoiding risks like pneumothorax, phrenic block, etc. For distal surgeries of short surgical duration, the IVRA and digital infiltration techniques can also be attractive options. Eliminating the tourniquet along with awake infiltration techniques can help fast track certain minor soft tissue procedures in nonoperating room settings.

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Conflict of interest

None.

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Section 2

Reconstructive Hand Surgery

Chapter 3

Flexor Tendon Injuries

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Additional information is available at the end of the chapter

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Abstract

Even though flexor tendon injury is common, much remains to be elucidated about the best way to facilitate the intrinsic healing process and to minimise scarring. This chapter will comprise flexor tendon anatomy, types of flexor tendon injury, modes of healing, molecular updates, repair techniques and post-operative rehabilitation.

Keywords: anatomy, flexor tendon injury, rehabilitation, tendon healing, molecular updates, complications

1. Introduction

Repair of the injured flexor tendon in the hand to achieve normal function remains a difficult task and is made even more so with complications such as repair rupture and adhesions. Controversy exists as to what post-operative rehabilitation protocols should be employed and is often based on surgeon experience or the hand therapy service. This chapter aims to focus on postoperative rehabilitation and future research trends. The reader is encouraged to seek alternative resources for more detail regarding flexor tendon anatomy, pathophysiology and repair techniques as these will be covered briefly in this chapter.

2. Flexor tendon anatomy

2.1. Flexor digitorum superficialis

Flexor digitorum superficialis (FDS) is responsible for flexion of the proximal interphalangeal (PIP) joint as well as contributing to the flexion of the metacarpophalangeal (MCP) joints. The muscle body has two heads of origin. The ulnar head arises from the anterior aspect of the

medial epicondyle, the ulnar collateral ligament of elbow, medial aspect of coronoid process, and the proximal ulna. The radial head originates from the proximal radius immediately distal to the insertion of supinator. At the mid-forearm, the FDS muscle belly divides into the superficial and deep layers. The superficial layer gives a tendon to the middle finger and ring finger, whilst the deep layer supplies the index and little fingers. Each tendon arises from a separate muscle belly allowing independent flexion of PIPJ of each finger except for LF which may have cross connection with FDS tendon of RF [1] as the FDS to the little finger may be hypoplastic or absent in some people [1].

At level of the proximal phalanx, FDS divides into two slips which pass on either side of the tendon of flexor digitorum profundus (FDP) forming "Camper's Chiasm" [1] and then unites to insert onto the volar surface of the middle phalanx. The blood supply to FDS is from both ulnar and radial arteries and its innervation is from the median Nerve which enters the muscle belly in proximal forearm.

2.2. Flexor digitorum profundus

Flexor digitorum profundus (FDP) is responsible for flexion of the distal interphalangeal (DIP) joints of the fingers as well as contributing to flexion of the PIP and MCP joints. It arises from volar and medial aspects of the proximal three quarters of the ulna and from the medial half of volar aspect of the interosseous membrane. It lies deep to FDS and adjacent to flexor pollicis longus (FPL) in the forearm. The Ulnar nerve travels distally in the forearm, loosely adhered to FDP, innervating the muscle-tendon units for the ring Finger and little fingers [1]. The anterior interosseous nerve (AIN) of the median nerve innervates the index and middle finger units.

Unlike FDS, the tendons of FDP usually arise from common muscle belly. Frequently however. The tendon to the index Finger arises from a separate muscle belly resulting in greater independence of flexion [1].

2.3. Flexor pollicis longus

Flexor pollicis longus (FPL) is responsible for flexion of the interphalangeal (IP) joint of the thumb. It arises from volar aspect of the middle one third of the radial shaft and from the interosseous membrane. It is located deep to the flexor carpi radials tendon and the radial artery at the level of the proximal wrist crease. FPL inserts onto the base of the distal phalanx of the thumb. The AIN innervates FPL in the proximal forearm and the blood supply is predominantly from the radial artery. All nine digital flexors enter the carpal tunnel with the four FDP tendons deep to FDS tendons of the little finger and index finger. FDS tendons of ring finger and middle finger lie superficially immediately ulnar to median nerve. The tendon of FPL lies deep and radial within the carpal tunnel, adjacent to scaphoid and trapezium.

2.4. Flexor sheath and pulleys

Proximal to MCP joints the flexor tendons enter the fibro-osseous flexor sheath. This tunnel functions to hold the tendons in close proximity to the phalanges to prevent "bowstringing" and to increase the efficiency of tendon glide [2]. The flexors heath tunnel is thickened in

certain areas called pulleys. The pulleys are either annular (A) or Cruciform (C) in shape and are numbered proximal to distal. The odd numbered pulleys A1, A3, A5 are over the MCP, PIP and DIP joints respectively [1, 3]. The A2 pulley overlies the proximal phalanx and the A4 pulley over the middle phalanx. The even numbered pulleys.

The cruciform pulleys are located proximal and distal to the PIP joints. Between the A2 and A3 pulleys is C1, between the A3 and A4 pulleys is C2, and the C3 pulley is located just proximal to the DIP joint between the A4 and A5 pulleys. The cruciform pulleys, in conjunction with A1, A3 and 5 permit compression of the flexor tendon without impingement during flexion and expansion of the tendon during extension [4]. The A2 and A4 pulleys have traditionally been deemed most important in preventing bowstringing and therefore most surgeons advocate their preservation, repair or reconstruction [4]. However, it has been demonstrated that partial distal excisions of 25% of the A2 pulley, up to 75% of the A4 pulley and 25% of combined A2 and A4 have no significant effect on digit range of motion or work of flexion [5, 6].

2.5. Tendon anatomy and nutrient supply

Tendons are collagen-based tissues that connect muscle to bone. They are composed primarily of Type I collagen, whereas the surrounding endotenon and epitenon are composed of Type III collagen [7]. The collagen itself is synthesised and secreted by tenocytes within the tendon and, once secreted, the collagen fibres arrange into triple helices to increase strength and stability.

Collagen fibre units are bound together by endotenon fascicles. These fascicles are bound in turn by the epitenon to form the whole tendon. Lymphatic, vascular and neural constituents are present within the endotenon to supply the fibroblasts whereas the epitenon contains the blood vessels and tracts for the lymphatics and nerves.

The tendon sheath is covered with synovial cells that lubricate the tendon to help gliding within the sheath. Outside of the hand, tendons are not typically enclosed within a sheath but are covered by paratenon that contains the vascular elements to supply both the endotenon and epitenon.

Both FDP and FDS tendons in the digits receive dual nutritional supply from vascular perfusion and synovial diffusion [7]. Vascular perfusion is provided by the vincula with each tendon having two: a vinculum longus and a vincula brevis. From proximal to distal, the first vinculum encountered is the vinculum longus superficialis (VLS), arising just proximal to Camper's chiasm and coming off the floor of the digital sheath of the proximal phalanx The vinculum brevis superficialis consists of small triangular mesenteries near the insertion of the FDS. The vinculum longus profundus arises from the superficialis at the level of the PIP joint. The vinculum brevis profundus arises near the insertion of the FDP. Each vinculum inserts on the dorsal aspect of the tendon, creating a richer blood supply on the dorsal side of the tendon. The vincula are important in the repair of injured tendons as they may hold the tendons out to length after injury. Therefore, one must be careful not to injure any maintained vincula while repairing an injured tendon, thereby decreasing the already tenuous blood supplyThe parietal paratenon allows for passive nutrition delivery to, and waste removal from, the tendons within the sheath by diffusion [8]. Nutrient supply is also directly from the bones of the fingers from the middle (FDS) and distal (FDP) phalanges.

2.6. Flexor zones

The volar surface of the hand is divided into five anatomic zones from distal to proximal. When describing a zone for an injury it is referred to by the zone that it would lie in the hand in the resting posture.

Zone 1 commences at the fingertip and ends at the insertion of FDS on the middle phalanx. It includes the C3 and A5 Pulleys and the FDP tendon.

Zone 2 is from the insertion of the FDS to the proximal aspect of the A1 pulley. It contains both FDS and FDP.

Zone 3 is from the proximal A1 pulley to the distal limit of the transverse carpal ligament (TCL).

Zone 4 is the area deep to the TCL i.e. the carpal tunnel.

Zone 5 is from the proximal edge of the TCL to the musculotendinous junction.

3. Types of flexor tendon injury

3.1. Patterns of injury

A sharp laceration to a flexor tendon is the most common cause of injury (for example from a knife or glass). It is rare for blunt injuries to divide the tendon but the significant crushing to the tendon can result in adhesions if not managed properly. Avulsion injuries are also common.

3.2. Lacerations

Lacerations may be complete or partial [2]. Lacerations within Zone 1 only involve FDP and those in Zone 2 usually involve both FDP and FDS tendons as well as any neuromuscular injury. Tears within the fibro-osseous sheath are more prone to restrictive adhesions than those within Zones 3–5. However, small lacerations in Zones 3–5 frequently involve multiple tendons and major neuromuscular structures

3.3. Avulsion injuries

Four factors determine the prognosis of avulsion injuries of flexor tendons: the extent of retraction of the proximal tendon, the remaining blood supply, the time interval between trauma and surgery, and the presence and size of any osseous fragments [2].

The FDP tendon is prone to avulsing from its insertion into the distal phalanx and is commonly called a “jersey injury” [9]. This occurs when the distal phalanx is extended at the DIP joint while the FDP is maximally contracted. This avulsion may involve a fragment of bone. Jersey finger most commonly affects the ring finger because it is the most proximal digit when the hand is flexed. Leddy and Packer have classified jersey injuries into Types I to III [9]. This classification has been modified by Smith who added a Type IV injury [10] and Al-Qattan who added Type V [11] (**Table 1**).

Type I	FDP and both vincula rupture with no fractures Tendon retracts into palm, presenting as a tender lump *Early operative repair necessary*
Type II	FDP ruptures but long vincula remains intact Avulsed FDP held at PIP joint here may be a small avulsed bony fragment *Repair required within 3 months*
Type III	FDP avulsion with a large bony fragment that gets caught at A4 Both vincula are preserved *No time limit for repair*
Type IV	FDP and bony fragment avulsion, with tendon avulsion from the bony fragment
Type Va	FDP and bony fragment avulsion, in association with fracture of the distal phalanx (extra-articular)
Type Vb	FDP and bony fragment avulsion, in association with fracture of the distal phalanx (intra-articular)

Table 1. Modified Leddy and Packer classification for FDP avulsion [9–11].

4. Tendon healing and latest molecular updates

Tendon healing undergoes overlapping inflammation, proliferation and remodelling [12] via two mechanisms -extrinsic and intrinsic [7]. Within the first week of injury, blood vessels within the tendon and tendon sheath form a thrombus at the injury site which acts to recruit vasodilators and proinflammatory cells [12]. These cells migrate to the injury site and help with removal of necrotic tissue, fibrin, clot and cellular debris through phagocytosis. Canine models have shown that angiogenic factors, such as vascular endothelial growth factor (VEGF), help initiate the vascular invasion to the site of injury [13].

In the third week, the tendon enters the proliferative stage whereby the fibroblasts rapidly proliferate, synthesis immature collagen in an unorganised manner, and assist with the production of extracellular matrix (ECM) [14]. This initial laid down collagen is type III collagen which is a weaker form of the type I collagen present in native tendons. The combination of type III collagen and previously initiated vascular network leads to scar formation within the tendon- this initially decreases its strength before the tendon enters the final stage of healing

At weeks six to eight, the remodelling stage predominates. Here type I collagen fibres are reorganised in a longitudinal manner along the long axis of the tendon with collagen fibrils crosslinking to one another to increase the strength of the tendon [14]. It is during this stage that adhesions between the tendon and its sheath become more apparent

4.1. Intrinsic healing

Intrinsic healing involves only the tenocytes (fibroblasts) within the tendon itself and depends on the migration and proliferation of cells from the epitenon and endotenon [7, 14]. Epitenon

tenocytes produce collagen earlier than those of the endotenon. Tenocytes of the endotenon produce large and more mature collagen than epitenon cells. In any event, both endotenon and epitenon tenocytes establish an extracellular matrix and internal neovascular network. Intrinsic healing results in improved biomechanics within the sheath, including tendon gliding. Movement of the tendon within the sheath improves synovial circulation and therefore the delivery of nutrients.

4.2. Extrinsic healing

Extrinsic healing involves the invasion of fibroblasts and inflammatory cells into the site of injury from the surrounding synovium, paratenon and tendon sheath [7, 14]. This produces scarring and peritendinous adhesions which may impair tendon movement, gliding and nutrition. It is thought that extrinsic healing predominates in the earlier stages of tendon healing. Extrinsic healing also predominates when tendons are immobilised after injury or repair. The extrinsic mechanism is activated earlier and is responsible for initial adhesions, the highly cellular collagen matrix and the high-water content of the injury site [7, 14]. The intrinsic mechanism then causes tenocytes from within the tendon to invade the defect and produce collagen which reorganises and aligns longitudinally to maintain fibrillar continuity and produce a healed tendon [15].

4.3. Research trends

Careful surgical technique and initiation of early motion after surgical repair of flexor tendon injuries have been the main strategies for decreasing tendon adhesions after surgical repair. Recent research has concentrated on improving the healing response within the tendon whilst decreasing the adhesion formation between the tendon and its sheath.

- **Transforming growth factor β (TGF-β):** Chang and colleagues [16, 17] have shown that the isoform TGF-β1, present in small amounts in native tendon and its surrounding sheath, increase in production after tendon transection and repair. TGF-β1 has been postulated to contribute to fibrosis and therefore scarring [18, 19]. Shah and colleagues showed that the neutralising antibody to TGF-β was able to control scarring in rat dermal wounds [18, 19]. Chang et al. added to this by demonstrating that these antibodies were able to increase the total range of motion after flexor tendon repair in a rabbit model [20]. However, suppression of TGF-β has been shown to decrease strength of tendon repair [21, 22].
- **Vascular endothelial growth factor (VEGF):** It is known that tenocytes secrete VEGF and is present in synovial fibroblasts [7]. After binding to its target, VEGF induces vasodilation [7]. VEGF mRNA levels are increased in flexor tendons after injury in the canine model [13]. These investigators are currently attempting to modify VEGF production to increase the vascular inflow to the blood supply of the healing flexor tendon.
- **Cell and molecular modulation:** Researchers have recently turned their attention to gene deletion strategies and gene therapy to modulate the healing process. Similar to inhibiting TGF-β, deletion of the TGF-β inducible early gene (*Tieg1*) resulted in decreased collagen I deposition in an *in vitro* model of tendon healing [23]. VEGF genes delivered by adeno-associated virus

(AAV) vectors in a chicken model demonstrated that healing strength was improved without increased adhesion formation [24]. Tang and colleagues [25] also used AAV vectors harbouring rat basic fibroblast growth factor (bFGF) to transfect chicken flexor tendons. Their results showed a moderate reduction in adhesions.

- **Tissue engineering:** Basile et al. [26] used a devitalised acellular allograft tendon containing recombinant AAV expressing growth and differentiation factor-5 as a delivery model. They were able to repopulate the graft, decrease scar tissue and enhance the gliding property relative to the control graft. Zhao et al. [27] demonstrated that lubricin combined with hyaluronic acid and bone marrow stromal cells stimulated with growth and differentiation factoir-5 can significantly improve gliding function in canine flexor models. However there were substantial decreases in repair strength compared to control In large animal models, synthetic membranes [28] and tissue-engineered synovial membranes [29] have been shown to decrease peritendinous adhesions.

5. History and examination

An accurate history and examination allows for planning of surgical approach. Though it is preferable for early tendon repair [30], immediate repair of a flexor tendon may be contraindicated in extensively contaminated wounds or those with substantial injury (involving two or more elements of skin, nerve, artery, vein, flexor tendons, extensor mechanism, bone or joint). Delayed presentation may also warrant surgical reconstruction of a flexor tendon due to proximal myostatic muscle-tendon retraction resulting inability to bring the proximal and distal stumps together.

It is important to perform a clinical examination of the traumatised hand before the administration of local anaesthesia to accurately identify and document neurologic or vascular injury [3]. Firstly, any volar laceration of the hand or wrist requires careful observation of the flexor cascade. In the normal cascade, each finger is slightly more flexed than the adjacent radial finger when the wrist is neutral or slightly extended.

To assess FDS, the FDP must be blocked from acting on the PIP joint. This is done by isolating the affected finger by holding all other fingers in extension and asking the patient to flex the PIP joint. By repeating the test against resistance, applied to the middle phalanx, partial lacerations of the tendon can be identified as it will elicit increased pain. FDP, responsible for flexion of the DIP, is tested in a similar manner to FDS. The middle phalanx is held in extension and the patient instructed to flex the DIP joint of each finger. Again, this can be done against resistance to identify partial tendon lacerations. FPL is tested by stabilising the proximal phalanx of the thumb and instructing the patient to flex the IP joint. However, a more reliable test of FPL function is using the 'O' sign where the patient is asked to make an O shape between their thumb and index finger. This O shape is only possible if the FPL is intact. This test is more reliable than asking the patient to flex the IP joint as there are trick movements that can cause a flicker of movement at the IP joint, causing diagnostic confusion.

6. Repair principles and techniques

The ideal method of flexor tendon repair should allow a healing response precisely at the tendon ends but not between the tendon and its surroundings, creates a repair site with minimal bulk and low friction, and places enough force across the repair to promote motion and remodelling [14].

The characteristics of an ideal tendon repair were described by Strickland [31] and confirmed by a large body of research data [14]. These are:

I. Core sutures easily placed in tendon

II. Secure knots

III. Smooth junctions

IV. Minimal gapping

V. Minimal interference with tendon vascularity

VI. Sufficient strength throughout healing to permit application of early motion stress.

VII. Motion at the repair site to increase the amount of collagen deposited at the site of injury and aid in the organisation of said deposition

VIII. Equal tension across all suture strands

Strength of the repair immediately post operatively is reliant on the suture and is therefore entirely responsible for the stability in early motion stress. Ideally, the suture material used should have high tensile strength, be inextensible, cause no tissue reaction and be easy to handle and knot [31]. Flexor tendon repairs consist of two parts. The core sutures and the epitendinous sutures.

Core sutures provide strength to the tendon repair. A 3/0 or 4/0 non-absorbable braided or monofilament suture are optimal for use as core suture [32]. The number of core sutures in the repair and the size of the suture is proportional to the strength of the repair. However, increasing the number of suture strands within the repair leads to increased bulk of the repair. Another factor in determining strength of the tendon repair is the grip of the core sutures. Increasing the grip of the core suture prevents the suture from pulling out of the tendon after repair [32].

The epitendinous sutures ensure smoothness of gliding and also increases the tensile strength of the repair. A locked running suture also reduces the rate of gap formation [32].

Large gaps in the tendon repair prevents healing with Gelberman et al. demonstrating that 3 mm is the maximum permissible gap to allow tendon healing [33].

Other factors include minimal handling to reduce adhesions and to avoid vascular interference to the repaired tendon [33]. Tendon lacerations less than 60% of the tendon diameter should not be repaired [34].

There are many techniques available for repairing the tendon and these have been described in detail elsewhere [35]. Four-strand techniques are generally recognised to be superior to

two-strand techniques. The choice of core suture can be made independent of the choice of epitendinous repair. There is little evidence to recommend one suture material over another. Steel and fibrewire have been shown to be stronger than nylon, Prolene and braided polyester, with no significant difference between the latter three [36].

7. Postoperative rehabilitation following flexor tendon repair

The surgeon must not only be aware of how to repair a lacerated flexor tendon but must also have an understanding of the postoperative rehabilitation regimen before consenting a patient for surgery. Failure to comply with rehabilitation may result in poor patient outcomes despite meticulous repair technique. Unfortunately, the best rehabilitation regimen remains to be elucidated. This is further convoluted by a paucity of well-designed randomised control trials with a Cochrane Collaboration analysis [37] being withdrawn in 2010 for being out of date and the most recent systematic review concluding that there is weak evidence supporting both early active motion protocols and combination protocols [38]. Therefore, it is imperative that the surgeon be aware of both current rehabilitation regimens as well as future directions. Current postoperative protocols for patients with flexor tendon injuries are immobilisation, early passive mobilisation and early active mobilisation [39] (**Figure 1**).

7.1. Immobilisation

Immobilisation may seem counterintuitive considering the plethora of studies showing the benefits of early mobilisation on the repair strength, tenocyte healing and formation of adhesions [14, 40–44]. However, there are certain situations in which immobilisation is preferable. These include patients who [39]:

- Are unwilling to adhere to strict early mobilisation protocols.
- Are unable to adhere to early mobilisation protocols such as children and those with cognitive deficits.
- Have injuries to other structures that could potentially be damaged by early mobilisation such as fractures, nerves and vessels.

O'Connell et al. followed 78 children (under the age of 16) for 24 months and found no benefits in early mobilisation protocols in children when compared to immobilisation [45]. However, immobilisation for more than four weeks resulted in functional deterioration of the repaired tendon [45]. Kato et al. found it difficult to encourage early active motion protocols in children aged less than six and found immobilisation for three to four weeks did not increase the incidence of tendon rupture or decrease function [46].

For the non-compliant adult patient, the protocol of Cifaldi, Collins and Schwarze may be used [39, 47]. It involves three to four weeks of immobilisation in a forearm-based dorsal splint or cast (20° wrist flexion, metacarpophalangeal (MP) joints in 50° flexion and the interphalangeal (IP) joints in full extension) followed by a weaning program (it may also be used in children) [47]. Weaning involves modifying the splint so that the wrist is in neutral and instructing the patient to remove the splint every hour and passively flexing

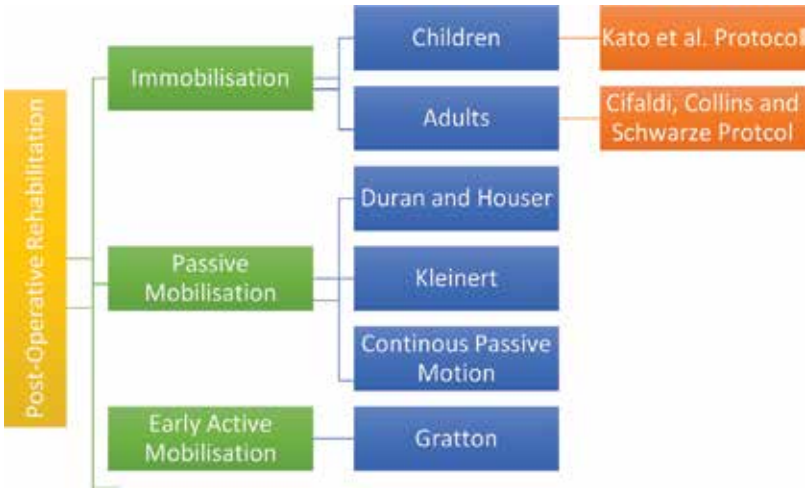

Figure 1. Summary of post-operative rehabilitation protocols.

and extending the injured digit for ten repetitions. Splint wear is then discontinued at six weeks (if flexion contractures of the wrist or fingers exist at this stage, a nocturnal volar splint holding the fingers in maximal comfortable extension may be worn). At this time differential FDS and FDP gliding exercises are performed every hour for ten repetitions. To isolate FDP gliding, both the MP and proximal interphalangeal (PIP) joints are held in extension, and the patient flexes the distal interphalangeal (DIP) joint. During this manoeuvre, the FDS tendon glide is prevented. The FDS tendon is isolated by holding all fingers in extension while the patient actively flexes the PIP joint of the affected finger. By holding the fingers in extension, the common muscle belly of the FDP is held to its full length, preventing it from assisting in flexion. At 8 weeks following surgery, sustained grip activities are added to the regimen. The resistance of these activities is gradually increased over the next four weeks. Heavy resistive exercises are avoided before 12 weeks because of the risk of tendon rupture.

7.2. Early passive mobilisation

Early passive mobilisation protocols are known to inhibit adhesion formation, promote intrinsic healing, and produce a stronger repair [42–44, 47–49]. Two well-known early passive mobilisation protocols are the Duran and Houser and the Kleinert protocols [39].

In the Duran and Houser protocol, the postoperative dorsal blocking splint holds the MP joints at 50° of flexion and the wrist at 20° of flexion. These investigators showed that three to five mm of tendon excursion was necessary to prevent firm tendon adhesions. The following regimen is followed twice daily. Using the opposite hand, the PIP and the DIP joints are brought from full flexion to full extension, with eight repetitions for each joint. Then, the patient performs eight repetitions of composite MP, PIP, and DIP flexion. This protocol continues through the fourth postoperative week. Approximately 5 weeks following surgery, the patients begin

active extension exercises with the use of a wristband. A rubber band is attached from the tip of the finger to the wristband, providing passive flexion and active extension. During this time, the patient also performs blocking and FDS gliding exercises. The late stage begins 8 weeks postoperatively. Progressive strength building is encouraged.

The Kleinert protocol uses a dorsal plaster splint immediately following surgery. This splint blocks the wrist and MP articulations in flexion. The wrist is placed at approximately 45° of flexion, and the MP joints rest at approximately 20° of flexion in the initial postoperative splint. The IP joints rest in neutral alignment in this initial splint. One week following surgery, the splint is replaced with a thermoplastic splint that maintains the same angles of flexion as the initial postoperative splint. The new splint allows for passive flexion of the digits and active extension of the digits against dynamic traction using rubber bands to facilitate the traction mechanism. The bands are placed on the volar aspect of the splint and directed toward the distal nail plate from just proximal to the wrist. Early passive ROM exercises are started within the confines of the dorsal splint. At one month following the patients remove the splint and began active flexion and extension exercises. However, patients wear the dorsal splint during periods of inactivity. About six weeks after the surgical repair, the dorsal splint is discontinued, and blocking exercises are started. Two months following the repair, resistive exercises are incorporated. Patients resume normal activities approximately three months following the surgical procedure. Problems that have arisen with the Kleinert protocol include flexion contractures of the PIP joint [50]. Treatment of contractures has consisted of continued intermittent splinting with the IP joints in neutral position [51]. In recent years, rubber band traction has been almost completely abandoned, largely because of the problems arising from the flexed resting position of the PIP joint [52]Continuous passive motion (CPM) uses devices that allow for joints to move through a predetermined arc of motion [39]. The aim is to increase the duration and repetition of exercises. Gelberman et al. [53] performed a randomised control comparing traditional early passive motion to CPM exercises and demonstrated that, at 6 months, the CPM group had significantly greater range of motion. However further research in evaluating the CPM following flexor tendon repair is lacking.

7.3. Early active mobilisation

An Early active mobilisation (EAM) protocol refers to active contraction of the repaired muscles [54, 55]. EAM promotes formation of large diameter fibrils and demonstrates the greatest cellular response to injury [48]. There are many different EAM regimens with some using a hinged dorsal blocking splint to assist with wrist synergistic motion whereas others use place and hold or active flexion [56, 57]. A well-known protocol is that by Gratton [58] who combined the Belfast and Sheffield practices [54]. The protocol is as follows:

- At postoperative day two to five, a thermoplastic dorsal blocking splint is fashioned with the wrist positioned in 20° of flexion and the MCP joints in 80° of flexion with the IP joints in full extension. If the patient has significant oedema, the active ROM exercises are delayed until day five. Oedema is treated with compression and elevation.

- If oedema is not significant, exercises begin with passive flexion of the digits with active extension to the constraints of the splint.
- Once these exercises are completed, the patient begins with active flexion exercises where a finger of the opposite hand is placed in the palm of the affected hand and the patient flexes the affected fingers against the contralateral fingers aiming to progress one finger width per week.
- By the end of the first week, the patient should have full passive flexion, full active extension and PIP active flexion to 30°.
- The splint is discontinued between weeks four and six (week four for patients with poor tendon gliding and six for those that have excellent ROM defined as full active fist at week two). The exercises at this time consist of passive ROM and active ROM.
- At week six, blocking exercises of the individual joint is commenced. At this stage, a splint may be needed to correct any flexion contractures.
- Strengthening begins 3 weeks after the dorsal block splint is discontinued. Strengthening progresses to allow patient to have full hand function by week 12.

None of the EAM protocols should be followed exactly- the surgeons and therapist must individualise treatment to patient circumstances [39, 59]. For example, advancement to the next phase of a protocol may need to be quicker or slower based on the level of oedema, passive versus active flexion lags, and adhesion formation [59]. Interestingly, the initiation of rehabilitation is a critical factor in successfully rehabilitating flexor tendon repairs. Initiating therapy by postoperative day five has been shown to decrease the rate of secondary procedures and decrease costs of treatment irrespective of whether or not a passive or active protocol is used [60].

8. Complications of primary repair

The most common complication of flexor tendon surgery are tendon adhesions which can limit the range of movement of the tendon. This is followed by re-rupture, joint contracture and triggering of the fingers. There is a 15–25% re-rupture rate after surgical repair [61]. Treatment of re-rupture of tendons is as follows [61]:

- if <1 cm of scar is present, resect the scar and perform primary repair
- if >1 cm of scar is present, perform tendon graft
- if the sheath is intact and allows passage of a paediatric urethral catheter or vascular dilator, perform primary tendon grafting
- if the sheath is collapsed, place Hunter rod and perform staged grafting

Rarer complications of flexor tendon injury repairs are Swan-neck deformity, Lumbrical plus finger, and Quadrigia effect.

9. Conclusions

Flexor tendon injury outcomes are unsurpassed when they are treated at an early stage. Good surgical technique is vital in to avoid rupture or adhesions. However, of equal importance is the preparation of the patient to expect an individualised long and complex rehabilitation programme.

Secondary reconstruction is complex and rarely results in the same level of function as a successful primary repair.

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Chapter 4

Current Treatment for Carpal Tunnel Syndrome

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Additional information is available at the end of the chapter

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Abstract

The combination of surgical procedure (open or endoscopic techniques), rehabilitation and antioxidant therapy (Alpha lipoic acid, curcumin) is superior to monotherapies in the prognosis and recovery of patients with this pathology. The prescription of these medications by their mechanisms of action should be allocated prior to decompression surgery and should continue receiving medication during the rehabilitation time. Clinical and electrophysiological follow-ups are required to verify the improvement.

Keywords: carpal tunnel syndrome, median nerve entrapment, compression neuropathy

1. Definition

The American Academy of Orthopedic Surgeons (AAOS) defines the carpal tunnel syndrome (CTS) as the most common form of entrapment neuropathy of the median nerve, and the syndrome affects 3.8% of the general population [1], with an incidence in both genders of 376 per 100,000 US habitants [2] combined and with a prevalence that usually varies in relation to the risk factors of a specific population; a study among poultry processing employees reported an estimated prevalence of 42%. CTS is one of the most common clinical problems encountered by hand surgeons. Although this syndrome is widely recognized, its etiology remains largely unclear [3].

2. Anatomy

The median nerve (MN) derives from the brachial plexus as a terminal branch of the medial and lateral cord. The fibers from the lateral cord (C6–7) provide sensitivity to the thumb, the index and the middle finger, as well as the motor fibers of the proximal muscles innervated by the median nerve (palmar muscles, pronator teres muscle). The medial cord (C8-T1) supplies most of the motor fibers to the distal muscles of the forearm and the hand, as well as the sensitivity to the external part of the ring finger. The MN descends through the arm without creating any branches until it reaches the forearm, just beneath the head of the pronator teres muscle, where its most important branch originates, the anterior interosseous nerve of the forearm. This nerve supplies the flexor pollicis longus, flexor digitorum profundus and pronator quadratus. Multiple muscular branches arise from the MN during its path, which supply the pronator teres, flexor carpi radialis, palmaris longus and flexor digitorum superficialis muscles. Proximal to the wrist and the carpal tunnel (CT), the palmar branch of the median nerve emerges and innervates the skin of the thenar eminence [4] (**Figure 1**).

In the palm of the hand, the MN sends a motor branch to the lumbricals of the index and the middle finger as well as a recurrent branch to innervate the muscles of the thenar eminence (abductor pollicis brevis, flexor pollicis brevis and opponens pollicis). The proper palmar

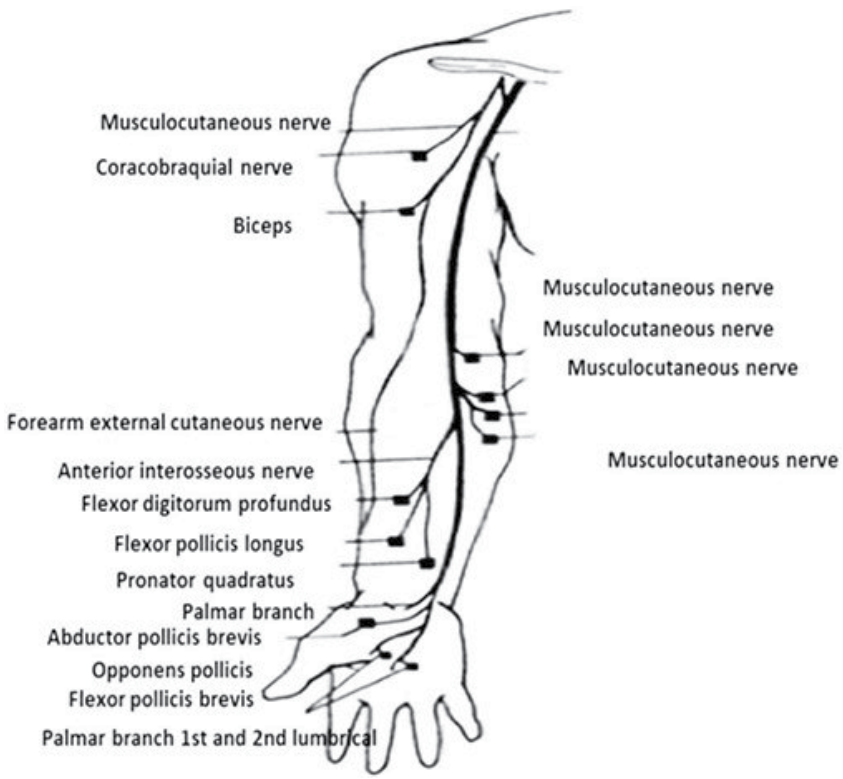

Figure 1. Median nerve pathway.

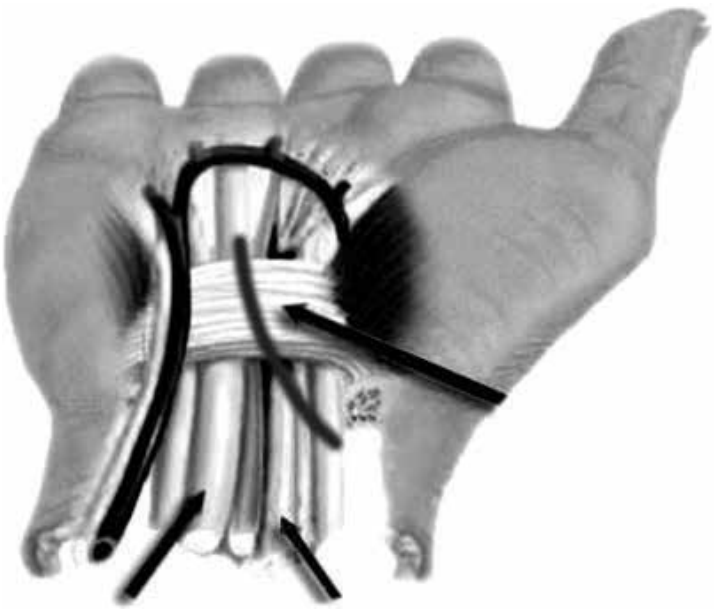

Figure 2. Anatomical elements involved in the procedure to open the transverse carpal ligament.

digital nerves are sensory fibers that supply the skin of the index and the middle fingers as well as the medial part of the ring finger and medial surface of the thumb [4] (**Figure 1**). There are multiple anatomical variants in the path and distribution of the MN, which could be present in up to 11% of the population. The most common variants are the presence of a medial residual artery, which is an embryological remnant that usually suffers a regression in the second trimester but could persist in 5% of the population, and it is also related to the presence of the CTS, and the anastomosis of Martin Gruber, which is a motor communication between the median and ulnar nerve at the forearm, which could have its origin from the union of the principal fibers of these nerves or in the anastomosis of the anterior interosseous nerve with the ulnar nerve and it is present in 5–10% of the population [5].

The carpal tunnel connects the anterior compartment of the forearm to the palm of the hand. It is delimited medially by the pisiform bone, laterally by the hamulus of the unciform bone, posteriorly by the scaphoid bone and the trapezoid bone and its roof by the transverse carpal ligament. It can be divided into three portions:

1. Proximal: it includes the volar carpal ligament, originated in the forearm.
2. Medial: the space comprehended between the pisiform bone and the unciform bone, and it has approximately 20 mm of width and a transversal area of 1.6 cm^2.
3. Distal: it corresponds to the origin of the palmar aponeurosis.

Many structures pass through the carpal tunnel, such as eight flexor tendons of the fingers (four superficialis and four profundus) and the flexor pollicis longus, and usually the localization of the median nerve is superficial to the tendons and medial to the flexor pollicis longus [4] (**Figure 2**).

3. Physiopathology of carpal tunnel syndrome

CTS is generally conceded as disarrangement caused by a decoupling of the size of the components of the carpal tunnel, and the space delimited by the fibrous and osseous structures. This is what conditions the compression of the median nerve, altering its irrigation. The compression of the components within the carpal tunnel induces venous congestion and epineural edema, consequently inducing fibroblast invasion in the affected tissue causing constriction and fibrosis of the endoneural compartment of the median nerve. The edema and the epineural and endoneural compression interrupt the axoplasmic flow of nutrients and ions and cause the median nerve to become enlarged [6].

Furthermore, the most common diagnosis is idiopathic CTS; nevertheless, recent studies that used magnetic resonance imaging (MRI), histological and biomechanical techniques have strongly suggested that abnormalities of the synovial tissue within the carpal tunnel are closely related to the development of idiopathic CTS, which means that subsynovial connective tissue may be predisposed to shear injury from activity done in 60° of wrist flexion [7].

4. New components to be highlighted in physiopathology of CTS

4.1. Effect of ischemia/reperfusion in the progression of the nervous injury in the carpal tunnel syndrome

The symptoms of CTS are caused by increased pressure within the carpal tunnel, and therefore, a decreased function of the median nerve. Nerve damage is attributed to restriction of blood flow in the endoneural capillary system, leading to alterations in the blood-nerve barrier structure and resulting in endoneural edema, venous congestion, ischemia and subsequent metabolic abnormalities. The ischemia-reperfusion injury of the median nerve results in oxidative stress and inflammation of the subsynovial connective tissue, and it has been proposed that this could have major contribution in the evolution of idiopathic CTS.

The intermittent compression of the vascular-nervous plexus due to a reduction of lumen of the carpal tunnel is one of the pathophysiological processes that is suspected to be the cause of development of CTS [1–6]. Nervous tissue has a very small capacity to tolerate ischemia (<20 min), which makes this tissue very vulnerable to be damaged [2–4, 6–10]; the component of narrowing of the carpal tunnel is intermittent, but persistent, which means that the injury is not presented acutely but rather progresses chronically [8–10].

The ischemia/reperfusion (I/R) phenomenon begins with an occlusion of arterial or venous blood flow to a tissue or an organ (ischemia); this interruption in the perfusion to the tissue will develop a direct injury in a limited area due to the ischemia, and this occurs in a specific time and depends on the affected organ (musculoskeletal, cardiac, renal, neuronal, adipose, tendinous, etc.). When the blood flow is restored (reperfusion), multiple local and systemic mechanisms will be activated in the affected area, which implies an increase of injury, known as I/R injury. The extension of this depend on the perfusion area of the affected vessel, the

time of ischemia and the repeated number of I/R events. Initially, it leads to an acute lesion proper of the phenomenon, then a major extension of the damage secondary to the repetition of I/R events, since in the CTS, it occurs in an intermittent and prolonged way [7, 10–12].

4.2. Components of the I/R injury in the carpal tunnel syndrome

There are multiple components in the I/R phenomenon [10, 12]; however, the most important components in the pathological development of the carpal tunnel are as follows: (1) increase in the cytosolic cations' concentration (change in the permeability of the membrane), (2) mitochondrial lesion (alteration of the ATP production and oxidative stress), (3) oxidative stress (production of reactive oxygen and nitrogen species coupled by disruption of redox reaction), (4) immunity-mediated lesion and (5) transcriptional reprogramming [11, 12] (**Figure 3**).

4.3. Alteration of the cellular membrane permeability

The activation of multiple transporters in the cellular membrane during the I/R phenomenon will lead to major changes (related to cellular microenvironment) in the calcium (Ca^{2+}) and

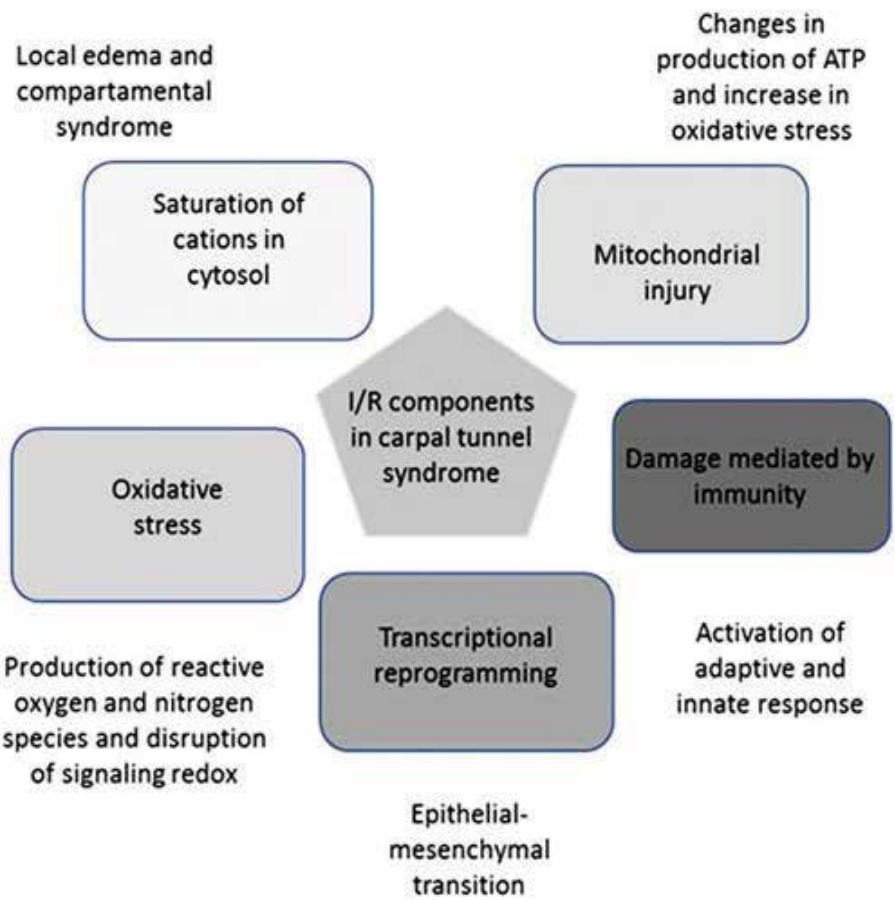

Figure 3. I/R components in carpal tunnel syndrome and pathophysiological events in the damage of local tissues.

sodium (Na^+) concentration. The movement of these cations through the membrane will be accompanied by water molecules that migrate from the extracellular space into intracellular and vice versa, making changes in the cellular volume leading to cellular and interstitial edema [9, 13, 14]. This edema expressed exclusively by the affected tissue will impact all the cellular groups in the adjacency such as nervous, adipose, muscular and endothelial cells from the structure of the carpal tunnel, and it can be accompanied by nervous or endothelial injury that will manifest with changes in the volumes of water in different compartments. These changes will produce secondary and interstitial compartment syndrome followed by edema, extended ischemia and local necrosis [9, 13–15].

4.4. Mitochondrial lesion

The mitochondria participate in multiple cellular activities such as ATP production and modulation of the redox state of the cell. The ATP production over the injury process is interrupted by a blocked complex in the respiratory chain (complexes III and IV), depletion of metabolic substrates (ADP, Pi, pyruvate, etc.) and high production of nitric oxide (NO^*) [15, 16]. The mitochondrial injury translates as a failure to adapt to the deprivation of oxygen and an OS overload to the enzymatic scavenger of the mitochondria in the affected cells by I/R phenomenon [16, 17]. If the lesion is substantial, it will be followed by fission processes of the mitochondria (fragmentation), loss of function and loss of the membrane potential [16, 17].

4.5. Oxidative stress and signaling redox

The blockage of the respiratory chain in the mitochondria increases the amount of oxidative stress (OS), leading to a production of reactive oxygen species (ROS) such as superoxide anion (O_2^*) and mainly, hydrogen peroxide (H_2O_2). The increase in the production of NO^* combined with the overproduction of ROS (in the phase of reperfusion) and the overload of scavenger systems in the mitochondria will magnify the production of reactive nitrogen species (RNS), primarily peroxynitrite ($ONOO^*$) [18–20].

The cells that present mitochondrial injury coupled with loss of mitochondrial membrane potential, "point of safe return" (PSR), will cause opening of mitochondrial permeability transitional pores (mPTP). The opening of these pores will liberate all mitochondrial ROS and RNS gathered in the matrix that will subsequently interact with cellular components (mostly lipids, proteins, nucleic acids), corrupting the function and triggering mechanisms of cellular apoptosis and autophagy [19–21].

The ROS and RNS production (OS) alters the function of the cell and make changes in the signaling redox of the adjacent survivor cells [22, 23], as some species of free radicals have the capacity to travel up to 400 nm in distance ($ONOO^*$) causing disturbance in the function and the configuration of membrane components and organelles in other healthy cells. By modifying redox signaling, the affected cells that survived the initial lesion can trigger a transcriptional reprogramming that will lead them to gene expressions of cellular injury such as pro-inflammatory cytokine receptors, making them susceptible to apoptosis induced by immunity-mediated cells [23].

4.6. Immunity-mediated lesion

The I/R lesion will activate three types of inflammatory responses such as sterile, adaptive and innate [11, 24]. The three kinds of immunity are activated by the OS activity generated in the mitochondria of affected cells by the initial lesion and the liberation of cellular material of necrotic cells (DNA, RNA, lysosomes, proteases, glucosidases, ATP, ADP, etc.), and this will secondarily result in the activation and differentiation of inflammatory cells, producing an adaptive response. In the lesion by IR, the toll-like receptors (TLRs) will be expressed in the cellular membrane, principally TLR2 and TLR4 [25]. Sterile immunity is characterized by the recruitment of neutrophils and macrophages and also by the production of pro-inflammatory and anti-inflammatory cytokines such as IL-1β, IL-6, TNF-α and IL-10 liberated by the damaged cells. The expression of pro-inflammatory cytokines will induce the local activation of the immune system causing necrosis of the previously injured cells and increasing the extension of the lesion [26].

The activation of multiple inflammatory systems due to the I/R phenomenon will involve a continuous inflammation that will persist while compression exists, increasing the damage; therefore, the recovery of this process will also depend on the chronicity of the injury. The transcriptional reprogramming of injured tissues will produce a change in some cellular groups, causing the expansion not only of acute but also of chronic and permanent lesion by the presence of fibrosis in the damaged area [27].

4.7. Transcriptional reprogramming

During the process of increased OS, disruption of signaling redox and inflammation will develop and result in transcriptional reprogramming, which involves a specific injured cell groups that will suffer a change in their structure and function, which is called epithelial mesenchymal transition. This produces cell mutation into pro-fibrotic phenotype cells, promoting permanent lesion of tissue and dysfunction of the limb [28, 29].

4.8. Clinical evaluation in CTS

In an individual with classical carpal tunnel syndrome, the most common symptom is pain accompanied by fingerprint weakness and numbness of the hand in the median nerve domain. The pain in CTS is characterized by two main pathophysiological processes: (1) acute ischemic pain due to compression and (2) chronic pain due to inflammation; the nervous tissue is the most susceptible tissue to the changes of oxygen and metabolic substrates [30]. The secondary lesion or death will manifest in sensation alterations and dysfunction, and the chronic lesion will lead to the formation of fibrosis and permanent lesion [31].

Two clinical provocation tests are useful to demonstrate severity and monitoring the progression. Phalen's test is applied by tapping over the median nerve as it passes through the carpal tunnel; a positive response is defined as a sensation of tingling in the distribution of the median nerve in the hand. Tinel's test is performed by hyperextending the wrist for 60 s; a positive response is defined as a sensation of tingling in the distribution of the median nerve in the hand [32].

The Boston Carpal Tunnel Questionnaire (BCTQ) is an easy, brief, self-administered questionnaire developed by Levine et al. for the assessment of symptom severity and functional status of patients with CTS. A validated version of the 11-item Boston Questionnaire for CTS (score range 11–55) is an evaluation instrument that was recognized as reproducible, valid, with internal consistency and able to respond to clinical changes, accepted in many countries for the assessment of severity of symptoms and functional status of patients, evaluating how the syndrome affects daily life [33] and the follow-up of progression (**Table 1**).

The severity of CTS is divided into three stages are as follows:

1. The symptoms presented during the first stage are as follows: waking up with the sensation of stiffness, numbness and weakness of the hand, perceiving the hand as swollen even though an increase in volume is not visible, pain with variable intensity that irradiates to the shoulder also called brachialgia paresthetica nocturna. The pain mitigates by shaking or flicking the hand.
2. In the second stage, the symptoms progress to being constant all day. Repeated hand or wrist motion and immobility of the hand for long periods of time may exacerbate the symptoms. At the moment of gripping objects, patients may also feel clumsiness or awkwardness.
3. The third stage is characterized by hypotrophy or atrophy of the thenar eminence, with a variable loss in the sensibility [31].

4.9. Electrophysiology

Nerve conduction studies (NCS) have to be performed immediately before the conservative treatment to follow-up progression and, in case surgery is required, evaluation before surgery and monitoring during recovery for at least after 3 months. Electrophysiology recordings from the median nerve could be analyzed in the context of Dumitru's reference values: distal sensory latency 3.0 ± 0.3 ms, distal sensory amplitude 15–50 μV, distal motor latency 4.2 ms and distal motor amplitude 13.2 ± 5 mV [34].

4.10. Treatments of CTS

Patients with mild or moderate CTS may first be offered conservative treatment. Options include splinting [35], corticosteroid therapy [36], physical therapy and therapeutic ultrasound [37, 38]. Patients with severe CTS and those whose symptoms have not improved after 4–6 months of conservative therapy should undergo surgical decompression. Endoscopic or open techniques are equally effective [39]. Clinical and neurophysiological improvements can be observed within the first 3 months of surgery, but up to 20% of patients may experience persistent postoperative sensory symptoms [40, 41].

4.11. Surgical procedure

The standard technique of open carpal tunnel release has proven to be effective and safe [42, 43]. The classical technique consists of a 7-cm curved incision just ulnar to the thenar crease and

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Boston Questionnaire Score

Name:

Evaluation date: ___/___/___ Surgery date: ___/___/

Hand: (___) Right (___) Left

The following questions refer to your symptoms within a typical period of 24 h during the last 2 weeks.

(1) How strong is the pain in your hand or wrist at night?	1. I feel no pain in hand or wrist at night. 2. Little pain 3. Moderate pain 4. Intense pain 5. Severe pain
(2) How many times did your hand or wrist pain wake you up in a typical night for the last 2 weeks?	1. Never 2. Once 3. Twice or thrice 4. Four to five times 5. More than five times
(3) Do you usually feel hand or wrist pain during the day?	1. I never feel pain during the day 2. I feel little pain during the day 3. I feel moderate pain during the day 4. I feel intense pain during the day 5. I feel severe pain during the day
(4) How often do you feel hand or wrist pain during the day?	1. Never 2. Once or twice a day 3. Three to five times a day 4. More than five times a day 5. Constant pain
(5) On average, how long do daytime pain episodes last?	1. I never feel pain during the day 2. Less than 10 min 3. From 10 to 60 min 4. More than 60 min 5. I feel constant pain during the day
(6) Do you feel your hand dormant (lost sensitiveness)?	1. No 2. I feel little dormancy 3. I feel moderate dormancy 4. I feel intense dormancy 5. I feel severe dormancy
(7) Do you feel weakness on your hand or wrist?	1. No weakness 2. Little weakness 3. Moderate weakness 4. Intense weakness 5. Severe weakness

Boston Questionnaire Score	
(8) Do you feel a tingling sensation on your hand?	1. No tingling sensation 2. Little tingling sensation 3. Moderate tingling sensation 4. Intense tingling sensation 5. Severe tingling sensation
(9) How strong is dormancy (lost sensitivity) or tingling sensation at night?	1. I never feel dormancy or tingling sensation at night 2. Little 3. Moderate 4. Intense 5. Severe
(10) How often did dormancy or tingling sensation wake you up during a typical night for the last 2 weeks?	1. Never 2. Once 3. Two to three times 4. Four to five times 5. More than five times
(11) How difficult do you feel in taking and using small objects such as keys or pens?	1. Not difficult 2. A little difficult 3. Moderately difficult 4. Very difficult 5. Severely difficult
Functional status of patients evaluates how the syndrome affects daily life	
In a typical day for the last 2 weeks, have your hand or wrist symptoms brought any difficulty in performing the activities listed below?	
Activity degree of difficulty	1. No difficulty 2. Little difficulty 3. Moderate difficulty 4. Intense difficulty 5. Cannot perform the activity at all due to hands and wrists symptoms
Writing	1 2 3 4 5
Buttoning clothes	1 2 3 4 5
Holding a book while reading	1 2 3 4 5
Holding the telephone hang	1 2 3 4 5
Housekeeping	1 2 3 4 5
Opening a glass vial cap	1 2 3 4 5
Carrying market bags	1 2 3 4 5
Bathing and dressing	1 2 3 4 5

Table 1. Boston questionnaire score (self-evaluation).

angulated over the flexion crease of the wrist in order to release the flexor retinaculum and antebrachial fascia under direct vision (**Figure 2**). Other options for an open technique consist of performing a short incision of 3 cm long in the mid-palm distal to the flexion crease of the wrist to dissect the flexor retinaculum under a deep plane to avoid subcutaneous fat and skin, then carefully sectioning the transverse carpal ligament using scissors. Although open release techniques have proven effectiveness and safety, some studies have associated minor complications with open release such as scar tenderness and pillar pain in the thenar and hypothenar eminences [43, 44]. There is no major advantage of short incision in the palm according to various studies; the major advantage is the size of the scar, which is shorter when compared to the classical incision. The time of return to work in comparing endoscopic, short incision and a classical incision do not show any significant differences between these techniques according to some studies [43, 44].

Endoscopic techniques to release the carpal tunnel, either single [44] or double-portal [45], reduce the morbidity and have a faster recovery period. Even though it offers theoretical advantages of reduced postoperative pain, quicker recovery of grip strength, fewer complications and faster return to everyday activities, endoscopic carpal tunnel release has not been widely adopted as the open techniques. It's worth pointing out that the risk of nerve injury increases with these types of techniques [46]. Actually, there is no sufficient consensus to support that the endoscopic technique overcomes open technique regarding clinical or electrophysiological recovery. The decision to perform endoscopic technique versus the classical or mini-open carpal tunnel release technique is most likely left to surgeons rather than patients, considering factors such as experience and resources.

4.12. From the pathophysiology to the therapeutic

The chronic lesion process will depend on the induction of I/R phenomenon on a repetitive pattern, in which the main pharmacological therapeutic target for regression or stopping the progression of the lesion would be OS modulation and inflammation.

The increase in OS production by the mitochondria in the I/R phenomenon is a key point to explore in the therapeutical approach to CTS, since the overproduction of ROS and RNS is involved in the activation of the inflammatory response and injury in the nervous cell [10, 16, 23]. The stimulation of the inflammation secondarily increases the production of OS; consequently, the therapy with antioxidants will be indicated in the acute and chronic phases of CTS.

The use of different antioxidants has been widely explored in the I/R phenomenon; however, there have only been made few investigations, which have had ambiguous results. From the point of view of evidence-based medicine, the use of antioxidants in the CTS treatment can produce a beneficial effect, accompanied by the decompression surgery, due to the impact in the pathophysiological process with the removal of the intermittent ischemic compression determined by the adjacent tissues in the walls and the reduction and modulation of the OS in the I/R phenomenon.

Nowadays, the most frequented phytodrugs that have shown positive results in CTS management and are focused on OS are as follows:

1. Alpha lipoic acid
2. Curcumin

4.12.1. Alpha lipoic acid

It is an essential substance for the function of different enzymatic components of the cells. It acts as a metal chelate, reducing free radicals, inflammation and modulating redox signaling. What stand out of this agent is the antioxidant, neuroprotective and neurotrophic properties, which exist in two isomeric forms such as R and S, where R plays a significant role in the pyruvate metabolism process and it is used in the mitochondria for the ATP generation. Its quality as metallic chelate is based on the interaction with divalent transitory metals (Mn, Cu^{+2}, Pb^{+2} and Zn^{+2}), and the ALA reduced form, which is dihydrolipoic acid (DHLA) (**Figure 4**), has the capacity to interact with Hg^{+2} and Fe^{+2}; the inflammatory modulation is through the nuclear factor kappa-B (NF-kB) path, it has been reported that degradation of IkB inhibitor can be suppressed by multiples mechanisms, supporting the reduction in pro-inflammatory cytokines production. The therapeutic doses that have been used for different pathologies range between 100 and 1200 mg/day and at least 2 weeks of treatment are needed for positive results and have been safely used for up to 4 years in clinical trials [47] (**Figure 4**).

4.12.2. Curcumin

It is an herbal polyphenol component with potent anti-inflammatory and antioxidant properties, extracted from the *Curcuma longa*, which has multiple therapeutic effects in different pathologies (cancer, autoimmunity, inflammation, metabolism, etc.). It has great distribution and favorable therapeutic range; however, its absorption rate and plasma half-life are short. The new formulation has given this phytodrug the capacity to achieve higher plasmatic concentrations, absorption and distribution, but more clinical studies are needed to be performed to confirm the information [48] (**Figure 5**).

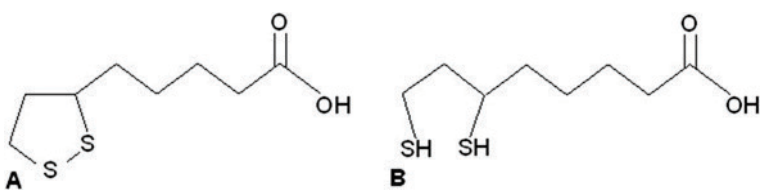

Figure 4. Structure of: (A) alpha lipoic acid structure and (B) dihydrolipoic acid.

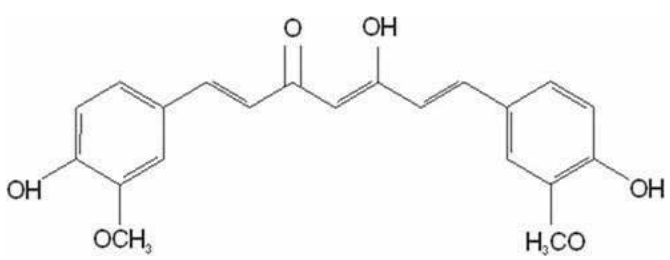

Figure 5. Curcumin structure.

4.12.2.1. Antioxidant mechanism of ALA and curcumin

The alpha lipoic acid and curcumin substances are OS scavengers through four mechanisms: (1) radical adduct formation (RAF), (2) hydrogen transfer (HT), (3) single electron transfer (SET) and (4) environments of different polarity. All these activities contribute to an exogenous reduction of ROS and RNS, modulating redox signaling, decreasing inflammation and mitochondrial lesion and modulating epithelial mesenchymal transition, which play a key role in the instauration of permanent lesion and progressive pain in the patient [49].

4.12.2.2. Clinical studies using antioxidants in CTS

Several investigators have reported favorable results with ALA as monotherapy or in combination with other antioxidants in CTS; the clinical trial conducted by Pajardi et al. [50] reported satisfactory clinical recovery from CTS with a combination of ALA, curcumin and vitamin B complex. Di Geronimo et al. [51] showed that clinical symptoms and neurophysiological outcomes were superior in a group that took a combination of ALA and gamma-linoleic acid compared with the group that took vitamin B complex. Notarnicola et al. [52] verified the efficiency of shock wave therapy versus nutraceutical therapy composed of ALA, linoleic acid, quercetin and Echinacea in CTS. Both groups showed clinical and electrodiagnostic improvement. Boriani et al. [53] recently published the effect of ALA in carpal tunnel syndrome, they used ALA as a monotherapy after surgery for 40 days, they showed good electrophysiological and clinical response; however, treatments with a longer period of time are necessary to observe the recovery of the nerve because during the first 30 days after surgery, the healing progress induces an immature and intense fibrosis; however, the recovery of the nerve does not occur.

Disclosure statement

The authors report no conflict of interests.

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Chapter 5

Dupuytren's Disease

Jeremiah D. Johnson, Colin Pavano and Craig Rodner

Additional information is available at the end of the chapter

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Abstract

Dupuytren's disease is a fibroproliferative disease affecting the palmar fascia of the hand and leading to flexion contractures of the digits. It was first described in Northern European populations and derived its namesake from Dr. Baron Dupuytren, a French surgeon, who was one of the first to lecture on the disease. The etiology of Dupuytren's disease is unclear but is likely influenced by both genetic and environmental factors. Older individuals and men are most at risk of developing the disease. Dupuytren's disease is a clinical diagnosis and patients often present with gradually worsening flexion contractures. Mild disease is usually observed, but surgical treatment is preferred for debilitating contractures. A variety of surgical techniques have been described involving either incising or excising diseased fascia. Overall, surgery is effective in correcting contractures and improving function, but despite successful treatment some patients still experience recurrence. More recently, collagenase injections and percutaneous procedures have been utilized to treat Dupuytren's disease and have yielded promising results in select patients.

Keywords: Dupuytren's disease, collagenase, flexion contractures, palmar fascia, limited fasciectomy, percutaneous needle fasciotomy, fibroproliferative disease

1. Introduction

Dupuytren's disease is one of the most common pathologies diagnosed in the field of hand surgery. It is a fibroproliferative disease of the palmar fascia characterized by flexion contractures of the digits and involves an abnormal production of type III collagen. The disease has a unique history with origins dating back to the Vikings and has been studied extensively since. However, despite ongoing research the etiology of the disease remains unclear but likely results from a complex interaction between genetics and environmental risk factors. Patients classically present with palpable palmar nodules and cords leading to gradual, progressive loss of function. The disease can potentially lead to debilitating flexion contractures of the digits

and affect activities of daily living. The disease is a clinical diagnosis and only requires further testing to exclude other pathologies. There is no cure for Dupuytren's disease and treatment methods remain palliative. Patients with mild disease can be observed for disease progression while patients with more severe disease may be treated with a variety of procedures or surgeries. Recently, clinical procedures including collagenase injections and percutaneous needle fasciotomy (PNF) have been utilized to successfully treat select patients. Still, surgery remains the preferred method of treating Dupuytren's contractures for most surgeons. A variety of surgical techniques have been described utilizing different types of incisions to either incise or excise disease fascia and correct contractures. Surgery has yielded successful outcomes in regaining extension of the involved digits and improving function of the hand, however, it is not without risks. Complications related to infection, wound healing, and neurovascular injuries have been reported. In addition, despite successful treatment following surgery some patients experience recurrence of their contracture. Further research has focused on methods of successfully treating Dupuytren's disease while reducing complications and recurrence. This chapter will provide a thorough description of Dupuytren disease from its history and pathophysiology to clinical management as well as highlight research related to patient outcomes.

2. History

Dupuytren's disease is a condition of the hand with a unique history. Its origin is linked to the Viking population and likely spread throughout Northern Europe as the Vikings conquered and acquired lands in the ninth through thirteenth century. For this reason, it has since been given the colloquial name, "Viking disease." The Viking's 300-year conquest lead to many settlements in which their descendants lived and bred with native populations, leading to the spread of Dupuytren's disease to many northern European nationalities [1, 2]. Naturally, as time progressed and more conquests occurred, the disease spread to the shores of North America, and is now found throughout the world.

Early evidence of diseases mimicking Dupuytren's has been noted in historical texts. The "Curse of the MacCrimmons" is a tale of seventeenth century Scotland in which Clan MacCrimmons was cursed with a "bent finger," leaving them unable to play their bagpipes [2, 3]. The Catholic Church sign of benediction has even been postulated to depict an early church priest with Dupuytren's disease [2, 4]. Whaley and Elliot describe early Icelandic stories of the twelfth and thirteenth century possibly describing accounts of Dupuytren's disease dating back to the ninth century and include the treatment of one case by a procedure resembling a palmar fasciotomy [5].

In 1831, the French surgeon and namesake to the disease Baron Guillaume Dupuytren gave a lecture on the "permanent retractions of the flexed fingers" [6]. Other surgeons have also been credited with describing conditions believed to be caused by Dupuytren's disease, including: Felix Platter in 1680, Henry Cline, Jr. in 1808, and Sir Astley Cooper in 1818 [2]. Elliot discusses a "Cline's contracture" as an earlier description of Dupuytren's disease [7]. MacFarlane reports Platter may have been the first to publish a description of Dupuytren's disease as early

as 1614 [2]. Despite possible earlier accounts, Dupuytren maintains the namesake of the disease to this day. Dupuytren finished his career as chief surgeon at the Hôtel de Dieu in Paris, and his name appears in at least 12 other diseases and instruments, cementing his legacy in the medical field [1].

Dupuytren's disease is often considered a "disease of the north," in to its northern European origins. Various studies into the prevalence of Dupuytren's disease demonstrate its northern roots, showing Norway, Scotland, and Iceland with some of the highest prevalence when compared to more southern nations [8–10]. Today, Dupuytren's disease can be seen in all types of patients; however, there are specific subsets of the population at in increased risk of developing the disease.

3. Epidemiology and risk factors

3.1. Prevalence

Dupuytren's disease is most prominent in Northern European white males, especially greater than 40 years old. A study in the Netherlands reports a prevalence as high as 22% in the general population [11]. The study also demonstrated a propensity for older populations with ages 50–55 displaying a 4.9% prevalence, while those 76–80 years old having a prevalence of 52.6%. Men were also affected disproportionately more than women (26.4 vs. 18.6%). The prevalence in the US has been shown to approach 7.3% when including self-reported symptoms [12]. Other epidemiological studies show a male to female ratio in the US of 1.7:1 which approaches 1:1 with increasing age [13]. Dupuytren's disease has been linked to both genetic and environmental factors, both of which contribute to the prevalence in patient populations throughout the world.

3.2. Genetics

The genetic component of Dupuytren's disease has been a topic of interest for many years. A study by Burge et al. found that the prevalence of Dupuytren's in Norwegian individuals over 60 years old reaches 30%, indicating a familial component in like populations [14]. They also suggested an autosomal dominant inheritance pattern with variable penetrance based on pedigree analysis. Multiple heritable patterns have been hypothesized, but there is no clear consensus on a mode of transmission. It is possible the disease does not carry a simple inheritance pattern, but rather follows a more complex method similar to heart disease and diabetes. Ling et al. performed a study examining the family members of patients with Dupuytren's disease, and found that 53% of men and 33% of women over the age of 60 in the family had signs of the disease [15]. In a clinical study, patients with a family history of Dupuytren's disease had a 6-year earlier onset of disease compared to patients without a family history [16]. There was also increased disease severity in terms of the number of affected digits and degree of contracture in patients with a family history. Both of these findings suggest patients with a family history of Dupuytren's disease develop a more severe and earlier onset of disease. Research has also investigated specific genes linked to the development of Dupuytren's, including the gene for TGF-β1. However, studies implicating TGF- β1 with

Dupuytren's disease have been inconclusive [17]. The genetic predisposition of Dupuytren's disease is complex and further research is needed to elicit a clear relationship between genetics and disease manifestation.

3.3. Environmental factors

Despite Dupuytren's genetic tendencies, multiple patients with no familial history of the disease are affected every year. Certain environmental factors have been associated with the development of Dupuytren's disease including smoking, alcohol use, diabetes, manual labor, and previous trauma. Hindocha et al. identified additional risk factors in developing Dupuytren's and included frozen shoulder, epilepsy, and a high lipid profile [16]. There are many environmental risk factors associated with Dupuytren's disease; however, smoking, alcohol, diabetes mellitus, and previous trauma are the most well-established factors cited in current literature.

3.3.1. Smoking

There is a high prevalence of patients with Dupuytren's disease who smoke cigarettes. A study found 76.5% of Dupuytren's patients were smokers, while only 37.2% of the control group were smokers [18]. Another study examined 222 patients undergoing surgery to treat a Dupuytren's contracture and found smoking was strongly associated with Dupuytren's requiring surgical intervention (OR 2.8, 95% CI 1.5–5.2) [19]. The pathogenesis of Dupuytren's disease from smoking is likely related to its impact on circulation. Cigarette smoking affects the small blood vessels causing microangiopathies, resulting in reduced blood flow to the distal extremities including the hands. The microvascular impairment from smoking is believed to contribute to the development of the Dupuytren's disease. Smoking induced hypoxia of distal extremities leads to PDGF release, triggering endothelial and fibroblast activation resulting in increased collagen synthesis [8]. These vascular changes associated with smoking and collagen synthesis may contribute to the pathogenesis of Dupuytren's. Overall, smoking is a modifiable risk factor that likely increases one's risk of developing Dupuytren's disease.

3.3.2. Alcohol

Though the role of alcohol is not clearly identified, it has been shown to be a risk factor for the development of Dupuytren's disease. One study suggested alcoholic consumption leads to impaired liver function, and in turn altered palmar fat composition which could as a trigger for developing Dupuytren's [20]. Heavy drinking was found to be more common in a study of Dupuytren's patients awaiting surgery, and another study reported alcohol was the second most important risk factor after age in developing the disease [20, 21]. Additional studies suggest that alcoholics have a higher prevalence of Dupuytren's disease compared to non-alcoholics (28 vs. 22%, respectively) [22]. Though associations have been documented, further research is needed in identifying the role of alcohol as a modifiable risk factor for developing Dupuytren's disease.

3.3.3. Diabetes mellitus

Multiple studies have identified a relationship between Dupuytren's disease and diabetes, but there is no clear evidence its pathophysiology. Noble et al. reported a 42% incidence of

Dupuytren's disease in adult diabetics and suggested Dupuytren's severity is usually more mild and affecting the middle finger in diabetics [23]. In addition, 13% of 134 patients with Dupuytren's disease were found to have elevated glucose levels, suggesting elevated blood glucose levels may influence development of Dupuytren's disease. In another study, the proportion of German diabetics with Dupuytren's disease was only slightly higher than that of the normal population (11% compared to about 7%) [24]. Another study however, showed the prevalence of the disease in diabetic patients was as high as 32% [25]. Though the prevalence of DD in diabetics has been studied in various subpopulations, a consensus on the relationship between Dupuytren's disease and diabetes mellitus has not been reached. Further research is needed to demonstrate a true mechanism of pathogenesis between Dupuytren's disease and diabetes.

3.3.4. Trauma

Dupuytren himself first proposed previous trauma as a risk factor for developing the disease [26]. The progression of Dupuytren's disease resembles normal physiologic healing and based on a patient's risk factors, trauma may initiate a cascade of events leading to an aberrant healing response. It is unclear whether the development of disease is related to a single injury or rather multiple insults over time, but studies have reported the development of Dupuytren's near the site of previous penetrating injuries [27]. Other studies have linked previous hand surgery such as carpal tunnel and trigger finger release to the development of Dupuytren's disease [22, 25, 28, 29].

3.3.5. Other risk factors

The association between Dupuytren's disease and epilepsy or anticonvulsants and has been reported. Lund et al. recorded a 50% prevalence of Dupuytren's in male patients with epilepsy and 25% in females [30]. Another study identified an overall prevalence of Dupuytren's disease in 37% of epileptics [31]. Critchley et al. reported a 56% incidence of Dupuytren's disease in chronic epileptic patients and an associated increased in disease with duration of epilepsy and possibly related to the administration of the anticonvulsant phenobarbital [32]. The effect of phenobarbital on DD was analyzed in a 2011 study, in which a dose-dependent fibrotic effect was seen with phenobarbital use. Though these studies demonstrate a relationship between epilepsy and phenobarbital use in DD, other studies have found no direct correlation to antiepileptic drugs [33, 34].

Manuel labor consisting of continued and repetitive hand use has also been proposed as a risk factor for developing Dupuytren's disease. Lucas et al. reported the effect of personal and occupation exposures on the development of the disease and reported men who developed the disease had the highest exposure to biomechanical, vibration, and manual work [35]. After adjusting for personal risk factors, manual labor and the handling of vibratory tools had the strongest association with Dupuytren's disease. Another study examining the effect of weekly hand transmitted vibration on the development of Dupuytren's disease concluded the risk of developing Dupuytren's contracture is more than double in men with increasing amounts of hand-transmitted vibration [34]. In addition, handwork for at least 30 years has also been reported as a possible risk factor in the development of Dupuytren's disease [35].

4. Anatomy and pathophysiology

4.1. Anatomy

A thorough understanding of anatomy is crucial to understanding Dupuytren's disease and its progression. Dupuytren's disease is a fibroproliferative disorder affecting the palmar aponeurosis, one of the three zones within the palmar fascial complex. The subcutaneous palmar aponeurosis is a continuation of the palmaris longus tendon, extending superficially to the palmaris brevis muscle and into the palmar surface. The palmar aponeurosis can be divided into three layers according to orientation: longitudinal, vertical, and transverse. The longitudinal fibers extend to the phalanges where they bifurcate and terminate as three separate insertions. The superficial layer inserts into the dermis and the deep layer inserts into the flexor and extensor mechanisms. The middle layer of the longitudinal fibers travel vertically next to the metacarpophalangeal (MCP) joint capsule forming spiral bands. Vertical fibers of the palmar aponeurosis include the superficial Grapow fibers, which anchor the skin to the aponeurosis, and the septa of Legueu and Juvara, which create fibro-osseous compartments to allow passage of flexor tendons, neurovascular bundles, and lumbrical muscles. Transverse fibers of the palmar aponeurosis include the transverse ligament and the natatory ligament.

Dupuytren's disease is characterized by the transformation of normal, palmar fascial bands into fibrotic, contracted tissue called cords. Different manifestations and stages of the disease are dependent on the anatomical bands that are affected. Early disease often affects the superficial Grapow fibers, forming thickened skin in the affected area. Pretendinous cords are the most common and result in skin dimpling and MCP contracture. The spiral cord consists of the middle pretendinous band, spiral band, lateral digital sheet, and Grayson ligament result in MCP and proximal interphalangeal (PIP) contracture and can also be accompanied with a medially displaced neurovascular bundle [36]. Other cords that can form include the central and lateral cords which can lead to a combination of PIP or distal inter-phalangeal (DIP) contractures while natatory cords can lead to web space contractures.

4.2. Pathophysiology

The pathophysiology of Dupuytren's is similar to the normal connective tissue healing process. However, there are aspects of the Dupuytren's process that differ and contribute to its pathogenesis. In Dupuytren's there is an increased number of myofibroblasts producing type 3 collagen, a change in biochemical composition of the fascia, as well as an abundance of cytokines and prostaglandins contribute to the pathogenesis. An immune mediated component of the disease has also been proposed and researched. Luck et al. describes three distinct microscopic phases of Dupuytren's disease [37]. The first stage is called the proliferative phase, and is characterized by increased fibroblast presence and proliferation in fascial bands, forming a nodule. On a cellular level, these nodules represent the accumulation of myofibroblasts, collagen, and extracellular matrix components within the palmar fascia, resulting in fibrotic, adherent lesions that decrease mobility of the joint. In this stage, there can be as high as a fortyfold increase in the amount of proliferating fibroblasts [38]. The active or "involutional" phase is dominated by myofibroblasts, which contain myofibrillar bundles in the cytoplasm,

allowing them to contract and draw tissue together. These muscle-like fibers align in the direction of stress [38, 39]. The residual phase is characterized by the disappearance of a nodule, and appearance of cords. The acellular cord causes shortening of the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joint and producing the classic contracted appearance of Dupuytren's disease. In the late phase, there is commonly impaired range of motion of the effected digit, and nerve damage and vascular insufficiency can occasionally be seen.

4.2.1. Myofibroblasts

Myofibroblasts are contractile cells that are α-smooth muscle actin positive and contribute to the contracture present in Dupuytren's disease. During the early phase of disease, myofibroblasts become densely packed in the palmar aponeurosis and produce an increased type 3/type 1 collagen ratio. The accumulation of fibroblasts and type III collagen creates the characteristic early nodule. This abundant type 3 collagen production is similar to connective tissue scar formation, and may or may not contribute to the pathophysiology of Dupuytren's disease [40]. The source of myofibroblasts in Dupuytren's disease remains unclear. Some argue that the fibroblast-myofibroblasts transformation is induced by local ischemia and hypoxia.

4.2.2. Biochemical changes

Biochemical changes to the palmar fascia may contribute to the development of Dupuytren's disease. These changes include increased glycosaminoglycan content, increased hydroxylysine content, and increased reducible crosslinks found in affected palmar aponeuroses [40]. Myofibroblasts found in the palmar fascia produce fibronectin, a glycoprotein thought to encourage cell-cell and cell-extracellular matrix adherence [41, 42]. This contributes to the thickening of the palmar fascia seen in the disease. Dermatan sulfate has been found to be two fold greater in tissue affecting by Dupuytren's and is known to affect collagen organization, deposition rate, and maintenance of fibers [43]. The same study also demonstrated an increased heparin sulfate composition in patients with Dupuytren's, which has been shown to play a role in cell recognition, adhesion, growth control, and angiogenesis. Research has also shown a change in the architecture of the palmar fascia in Dupuytren's patients. Compared to normal fascia, fascia in Dupuytren's patients has more hydroxyl-lysino-hydroxy-norleucine crosslinking which was absent in normal tissue [44, 45]. It is unclear how this increased crosslinking component contributes to the severity of disease.

4.2.3. Cytokines and prostaglandins

Many studies have pointed to the role of growth factors and cytokines in the pathogenesis of Dupuytren's disease. An increased production of IL-1α and IL-1β is seen in Dupuytren's palmar fascia. [62] These cytokines are involved in proinflammatory processes including local fibroblast proliferation, which can potentially contribute to the active stage of Dupuytren's disease [46]. There is also an increase in bFGF which contributes to fibroblast growth and proliferation, and increased TGF-β, which contributes to collagen synthesis and fibroblast chemotaxis [46]. The study concluded that increased release of cytokines and growth factors relative to normal reparative tissue may suggest a locally driven fibroblast proliferation contributing

to the development of Dupuytren's disease. Research has also demonstrated prostaglandins PGE2 and PGF2α play a role in contractility of smooth muscle associated with myofibroblasts [44, 47]. This contractile influence on myofibroblasts is thought to contribute to the contraction of tissue late in the disease. The source of these prostaglandins are possibly from microcirculation and perinodular fat, as nodules are highly vascularized and fatty.

4.2.4. Immune mediated

Studies have suggested an immune mediated response in the pathophysiology of Dupuytren's disease. Mayerl et al. describe abundant accumulation of immune cells in Dupuytren's tissue, including mononuclear CD3+, CD4+ > CD8+, and primarily a Th1 mediated response [39]. These clusters of immune cells were found around blood vessels in the area, suggesting the fibroproliferation exists in Dupuytren's may be due to microvascular damage mediated by the immune system. Further research is required to determine the relationship of Dupuytren's to an immune mediated response.

5. Clinical presentation and physical exam

A thorough history and physical exam is necessary to accurately diagnose Dupuytren's disease, which has a classic presentation. It is important to assess risk factors including family history, northern European descent, smoking, alcohol use, history of diabetes mellitus, or previous trauma to the hand in order to gain a broader picture of the patients' presentation. In many cases, disease is bilateral with one hand affected more than the other so it important to evaluate both hand. The ring finger is the most commonly affected digit followed by the small, middle, index, and thumb [48]. Other fibroproliferative diseases have been associated with Dupuytren's disease and include Garrod's nodes, Ledderhose disease, and Peyronie's disease. Garrod's nodes, also called knuckle pads, can be visualized on the dorsal PIP joints and are subcutaneous nodules that histologically portray myofibroblasts proliferation. Ledderhose is a fibrosis of the plantar fascia and Peyronie's disease is an inflammation and scarring of the tunica albuginea of the penis. Rayan et al. described three phases to Dupuytren's disease clinical presentation: the early, intermediate, and late phases [49]. These three phases provide a good framework to assess the patient's disease status, and each phase is characterized by distinct aspects of the disease.

5.1. Early phase

Early disease is characterized by skin dimpling, puckering, and pitting, usually on the medial aspect of the palm. These changes can lead to the patient seeking medical attention, however are also easily ignored by some patients. A Dupuytren's patient population study suggests that approximately 11% of Dupuytren's disease patients seek attention from their physician with a chief complaint of skin changes on the palm [12]. Physical exam during the early phase of disease can confirm these skin changes upon inspection. Pitting of skin on the medial palm of the hand is a good indication of developing Dupuytren's disease. Palpation reveals thickening and

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dimpling of the skin around the effected joint. The underlying fat on the medial palm becomes fibrotic near the distal palmar crease. Active range of motion (ROM) and strength testing in early disease will reveal no limitations, though more severe skin adhesions can lead to a slight decrease in mobility and function of the affected digits in some patients.

5.2. Intermediate phase

The appearance of Dupuytren's nodules and cords signifies the intermediate phase of disease. Nodule formation is often one of the first patient complaints and occurs during intermediate stages of the disease. Approximately 42% of patients with Dupuytren's disease present to the office due to a nodule [12]. Nodules most commonly form proximal to the palmar crease overlying the metacarpophalangeal joint of the affected digit, and encompass the superficial layers of the palmar and digital fascia. Sometimes digital nodules are seen at the base of the proximal interphalangeal joint. Though often painless, larger nodules can cause pain when they exert pressure on underlying flexor tendons. Painful, chronic nodules are more indicative of intrinsic joint disease and rheumatoid arthritis, and must be differentiated from a Dupuytren's disease nodule. After the appearance of a nodule, a pathologic cord may form within the palmar fascia. Approximately 12% of patients seek will seek care following development of a cord [12]. Nodules often regresses, but in some cases can be present simultaneously with Dupuytren's cords. Initial cords are often unnoticeable and blend in with the underlying connective tissue, but over time, they become thick and resemble subcutaneous tendon-like structures upon inspection (**Figure 1**). Palpation reveals an immobile, thickened cord. Cord formation is extremely variable in terms of location. The most common cords arise in the palm, and include peritendinous, natatory, and vertical cords arising from their respective bands in the palmar

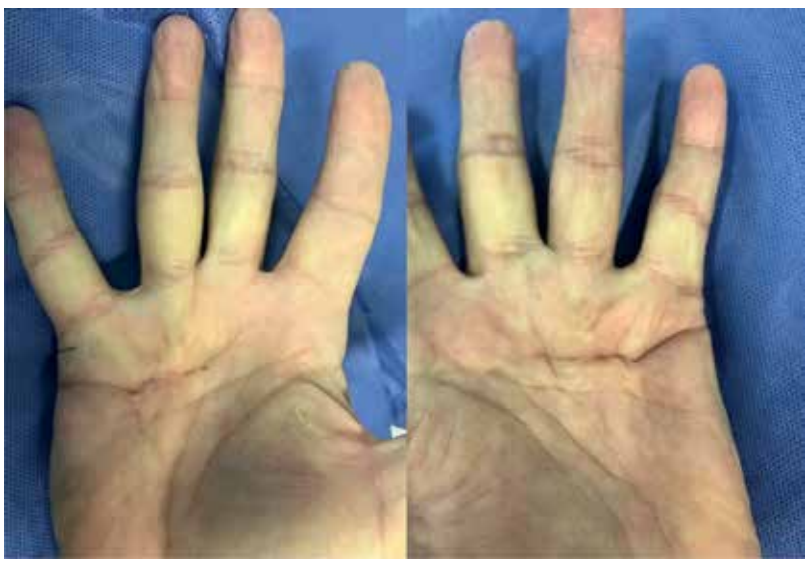

Figure 1. A patient with Dupuytren's cords leading to contractures and affecting the bilateral small and ring fingers.

fascia. Digital cords frequently seen include central and spiral cords. Active and passive ROM testing during the intermediate disease will often reveal no limitations in patients with nodules, but as cords form patients will begin to lose extension of the involved joint.

5.3. Late phase

Late disease is defined by contraction of cords and the classic "bent finger" appearance of Dupuytren's disease (**Figure 2**). Approximately 10% of Dupuytren's disease patients will present during the late stage of the disease complaining of a permanent bent finger [12]. Contracture of the MCP joint often occurs before the PCP joint. Contractures often lead to difficulties in activates of daily living and patients will report difficulties with chores, washing, putting a hand in a pocket, and handshakes. Inspection and palpation will reveal a contracted, fibrotic cord. Both active and passive finger extension of the effected finger will likely be impaired, the extent of which is determined by severity of disease. Pain with ROM is rarely reported and if present should prompt further evaluation. The table top test was described in 1982 by Hueston and is specific to a Dupuytren's diagnosis and has been used to stage disease progression [50]. The test involves placing the patient's hand on a tabletop with the palmar side down. The test is positive if the patient cannot flatten the hand against the table and is indicative of the late phase of the disease.

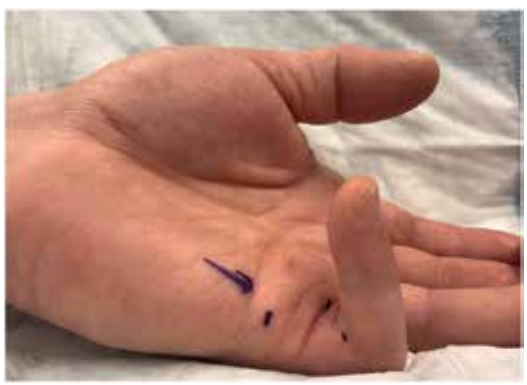

Figure 2. A patient with Dupuytren's PIP joint contracture with a flexion contracture of approximately 100°.

Tubiana stage	Degrees of extension deficit
0	0, No disease
N	0, Nodules present
I	1–45
II	46–90
III	91–135
IV	>136

Table 1. Tubiana staging system based on a digit's total extension deficit [51, 52].

The degree of extension deficit is taken into account when staging Dupuytren's disease. The adapted Tubiana staging system is the most common method of classifying the progression of Dupuytren's disease (**Table 1**) [51, 52].

6. Diagnosis

Dupuytren's disease is a clinical diagnoses based on a patient's history and physical exam. Its hallmark features consist of an indolent, progressive course characterized by palmar skin changes, painless nodules, and fibrotic cords leading to flexion contractures of the digits. Patients presenting with later findings of the disease consisting of fibrotic cords and contracted digits are more clearly diagnosed. Conversely, patients presenting with earlier features of Dupuytren's disease such as painless nodules may not be as easily distinguished from other diseases. Stenosing tenosynovitis, also known as a trigger finger, and soft tissue tumors may be mistaken for Dupuytren's disease. Stenosing tenosynovitis can be differentiated from Dupuytren's by tenderness over the A1 pulley with symptomatic locking or triggering of the digit and often no ROM deficit. Soft tissue masses typically do not present with skin thickening and pitting as seen in Dupuytren's disease. In addition, Dupuytren's nodules are often fixed to the skin and palmar fascia. Early Dupuytren's disease may be difficult to distinguish from diabetic cheiroarthropathy, however, involvement of multiple digits and a waxy appearance of the skin are clues to distinguish diabetic cheiroarthropathy. Other pathologies that may present with some features similar to Dupuytren's disease include: ulnar claw, rheumatoid arthritis, Volkmann's contracture, and camptodactyly. Radiographs should be considered in patients presenting with a history of trauma to rule out a fracture or dislocation. Further diagnostic imaging such as MRI may be considered in special cases to rule out suspicion of other disease processes, but is not required to diagnose Dupuytren's disease. A thorough history and physical exam is key to accurately diagnosing Dupuytren's disease.

7. Nonoperative treatment

Despite the recent advances in understanding the pathophysiology of Dupuytren's disease the treatment options remain palliative and not curative. Non-operative treatment is recommended in patients with isolated disease without contractures and in patients with mild contractures without significant interference with activities of daily living. Observation is a reasonable non-operative option for many patients with early disease and minimal symptoms. Studies have estimated about 50.8% of patients with palpable nodules will progress to developing cords after 8 years from diagnosis, and of these only 17% will develop contractures meeting criteria for surgical intervention [53].

Surgery is the mainstay treatment for Dupuytren's contractures. However, non-operative interventions continue to be pursued as an alternative option to surgical intervention. Splinting and physical therapy have mostly been utilized as a post-operative intervention to prevent recurrence. Critics of splinting and physical therapy often express concern it may worsen the contracture if the contractile tissue is not first removed. In vitro studies have reported

mechanical loads increase TGF-beta expression and thus enhance fibroblast contraction [54]. Few clinical studies have investigated orthosis or therapy as a non-operative intervention. Larocerie-Salgado et al. reported patients with mild PIP joint contractures had an average improvement of 14.6° (SD 5.1°) after wearing a volar hand-based extension splint at night and utilizing hand exercises and massage [55]. Another study comparing tension and compression orthosis worn for 20 hours per day reported significant improvement in the total active extension (TAE) of a digit in both groups compared to baseline TAE [56]. Overall there is minimal evidence regarding therapy and orthosis usage. There may be some benefit in preventing progression of a contracture in an isolated digit, but the possible benefit may be minimal and outweighed by interference of the splint and necessity for prolonged periods of daily use.

Intralesional triamcinolone injections have also been proposed to deter the progression of Dupuytren's disease. Steroid treatment in Dupuytren's is linked to its effectiveness in reducing hypertrophic scars and keloids by degrading insoluble collagen. Ketchum and Donahue reported resolution of Dupuytren nodules in patients with mild disease (<15° joint contracture) after an average of three 60-120 mg triamcinolone injections spaced out over 6 weeks [57]. However, 50% experienced recurrence of disease and either underwent further injections or surgery. Other studies have utilized triamcinolone in patients with nodules but without flexion contractures and have demonstrated better outcomes with only a 6% recurrence at 5 years [58]. The high recurrence rates and complications including skin atrophy, transient erythema, depigmentation and tendon rupture have minimized the use of steroid injections in Dupuytren's disease [57, 58].

Non-operative treatment has recently expanded to include office-based procedures to provide patients with Dupuytren's contractures an alternative to surgery or treat patients unable to tolerate surgery. Percutaneous needle fasciotomy and collagenase injections are two clinic procedures that have recently gained popularity. Zhao et al. reported these two minimally invasive techniques comprised 14% of all procedures for Dupuytren's in 2007, but have more recently risen to 39% of all procedures [59].

7.1. Percutaneous needle fasciotomy

Percutaneous needle fasciotomy (PNF) utilizes a 25-gauge needle as a scalpel to incise the contracted cord at different levels while the digit is manually straightened. Prior to the procedure the dermis is injected with a local anesthetic to reduce pain and is followed by range of motion (ROM) exercises aimed at preventing recurrence of the cord. As described by Eaton, needle fasciotomy has four requirements including a contracture caused by a palpable cord, redundant skin, and a cooperative patient [60]. The benefits of the procedure include a low complication rate, early return of motion, and avoidance of surgery. Zhou et al. reported a complication rate of 5.2% after fasciotomy compared with 24.3% in a group undergoing limited fasciectomy [61]. However, PNF had a higher rate of recurrence at long-term follow-up. Van Rijssen et al. have reported recurrence rates following PNF as high as 63% at 3 years and 84.9% at 5-year follow-up [62]. Patients older than 75 years old with mild disease had the lowest rate of recurrence at 5 years. In general, PNF is a reasonable option for older patients who have developed a mild contracture due to a palpable cord and are well-informed of the recurrence risk but prefer a minimally invasive option.

7.2. Collagenase injections

Collagenase injections were first approved for Dupuytren's contractures in 2010 by the United States Food and Drug Administration and are currently approved for the treatment of two Dupuytren's contracted joints in the same hand. Collagenase injections deliver an enzyme isolated from *Clostridium histolyticum* which is responsible for lysing the collagen in a contracted cord. After injection, the patient returns within 1–3 days for manipulation to straighten the digit. Hurst et al. reported good results following up to 3 injections with 64% of patients experiencing 0–5° of full flexion with no recurrence 90 days after treatment [63]. Common adverse events of collagenase included swelling, pain, bruising, tenderness, and pruritis. Complications related to tendon ruptures, skin atrophy, and complex regional pain syndrome (CRPS) are rare and have been reported in less 1% of patients [63, 64]. Recurrence rates following collagenase have been closely studied as well. Van beek et al. reported 2-year recurrence rates (>20-degree worsening) following one or more injections were 28.2 and 62.1% for MCP and PIP joints, respectively [65]. Peimer et al. reported 47% of successfully treated patients experience recurrence (>20° worsening) within 5-years following collagenase injections with PIP joints having a higher degree of recurrence at 66 versus 39% among MCP joints [64]. Collagenase injections provide a good treatment option for patients with a palpable cord causing a contracture.

8. Operative treatment

Operative treatment of Dupuytren's disease is offered in patient with contractures of >30° at the MCP joint and any functionally bothersome PIP contracture. The goal of surgical treatment is to return full extension of the involved digits via various surgical techniques involving either incising or excising the diseased fascia. Dupuytren originally described an open palmar fasciotomy technique in 1831 and this was later popularized in 1964 by McCash et al. as the open-palm technique [6, 66]. The open-palm technique involved a transverse incision across the distal palmar crease followed by incising any Dupuytren cords. Multiple surgical methods have since been described and include open fasciotomy, segmental fasciectomy, limited fasciectomy, and dermofasciectomy. These techniques range from being minimally invasive to radical excision of the diseased tissue. It is important to consider the severity of contractures, extent of correction, and risk factors for recurrence in addition to protecting soft tissues when choosing the optimal surgical treatment. Surgery is ultimately an elective form of treatment and should prompt a conversation with patients regarding the risk and benefits of surgery as well as their functional goals.

8.1. Soft tissues

Proper handling of soft tissue is a key principle of surgical treatment of Dupuytren's contractures. Adequate exposure of the cord must be balanced with protecting the neurovascular bundles, providing adequate wound coverage, limiting the risk of skin necrosis, and avoiding secondary contractures from longitudinal scarring. Multiple skin incisions have been described to address these issues and include: transverse incisions in the palm and digit, a

Bruner incision, a Bruner incision with V-Y advancement flaps, curved incisions, and a longitudinal incision closed with z-plasties (**Figure 3**). Midline incisions provide the benefit of potentially avoiding the neurovascular bundles, however, Dupuytren's disease distorts regular anatomy and can tether the cord toward the midline. Curved or zig-zag incisions help avoid secondary contractures from longitudinal scars. Despite the many variations in incisions, the Bruner incision seems to be the most commonly used.

Skin closure is another important aspect of treating Dupuytren's disease. The open-palm technique described by McCash left the skin incisions open to decrease hematoma formation and allowed for secondary healing with good results [66]. Today, most incisions are closed primarily, however, increased skin tension after closure has been correlated with elevated recurrence rate (**Figure 4**). Citron and Hearnden randomized patients undergoing fasciotomies and reported a 50% recurrence rate in the group with transverse incisions closed primarily compared with a 15% rate in patients with longitudinal incisions with a z-plasty closure [67]. Another study compared Bruner's incision with direct closure to a longitudinal incision with a z-plasty closure for fasciectomy and reported no difference between the two methods [68]. Special attention should be given to skin tension during closure and a transpositional flap such as a z-plasty should be utilized if needed.

8.2. Open fasciotomy

Open fasciotomy includes a variety of surgical techniques for treating Dupuytren's contractures by incising the contracted cord without removing the diseased fascia. Many of the modern fasciotomy techniques are modifications of the methods originally described by Dupuytren and

Figure 3. The figure outlines types of incisions and closures options for treating Dupuytren's disease. Thumb: longitudinal incision with z-plasty closure. Index finger: Brunner incision. Middle finger: curvilinear incision. Index finger: V-Y incision. Small finger: transverse incisions utilized in the McCash open palm technique.

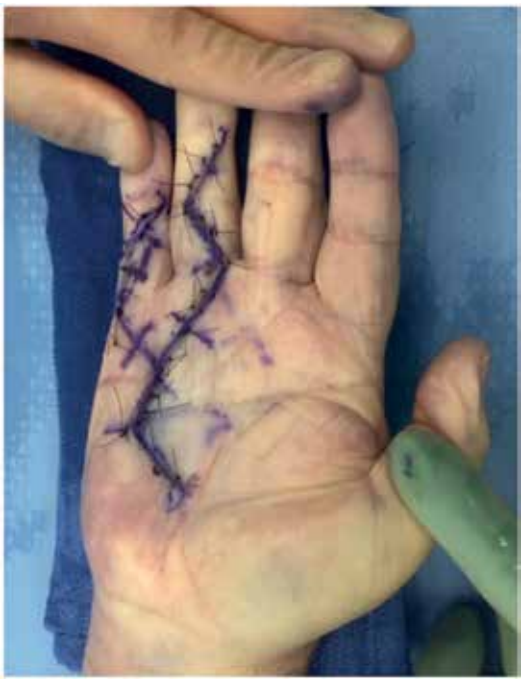

Figure 4. A post-operative image of a patient after a limited fasciectomy for a Dupuytren's contracture of the small and ring finger. The Brunner's incision was closed primarily with nylon sutures.

McCash et al. [6, 66]. Various types of incisions may be utilized to access the cord, but once the cord is visualized and incised, the digit is extended until straight. In some cases, additional incisions may be required at different levels along the cord in order to fully extend the digit. Unlike PNF, an open fasciotomy procedure provides the benefit of direct visualization to protect neurovascular structures and can often be accomplished through small incisions. In addition, it minimizes the potential morbidity sometimes seen in other techniques which excise the diseased fascia. Still, excision techniques are often preferred over an open fasciotomy for their ability to remove the diseased fascia which may aid in preventing regrowth of the cord and recurrence. This may be particularly true in patients with severe contractures. Stewart et al. retrospectively reviewed a series of patients who had open fasciotomies and reported a reoperation rate of 13.2% at 46 months with patients who initially required three level fasciotomies having worse recurrence [69]. Another study assessed 16 patients with Tubiana stage III and IV contractures and reported a higher recurrence rate of 37.5% at 5–8 year follow-up [70].

8.3. Fasciectomy

Fasciectomy is the most common surgical treatment for Dupuytren's contractures. The technique is based on the concept that the remaining diseased fascia may proliferate and lead to recurrence. Multiple methods of fasciectomies have been described and range from a segmental fasciectomy, where a portion of the fascial cord is removed, to a radical fasciectomy where skin and diseased fascia are excised. A segmental fasciectomy is usually completed through small incisions where segments of the cord are excised until the finger straightens. No attempt

is made to removal the complete cord. The most widely used technique is a limited fasciectomy and is considered the gold standard in the operative treatment of Dupuytren's contractures (**Figure 5**). The technique involves carefully exposing the diseased fascia from its proximal to distal end and excising it from the surrounding soft tissue (**Figure 6**). It differs from a radical fasciectomy by removing only the diseased fascia and leaving normal fascia, subcutaneous tissue, and the dermis intact. Radical fasciectomy advocated by McIndie and Beare involved extensive removal of nearby tissue and skin and required a skin graft [71]. However, the technique largely fell out of favor due to a higher complication rate without a reduction in recurrence. A dermatofasciectomy is similar to a limited fasciectomy but involves excising the overlying skin and often requires a skin graft for coverage. Advocates of this technique report disease-forming cells may be left in the overlying soft tissue leading to recurrence and selectively use it in patients at higher risk for recurrence. Despite the variety of techniques, fasciectomies require meticulous dissection to avoid injuring neurovascular bundles which may be displaced by a contracted cord. In addition, maintaining hemostasis is important to prevent hematoma formation which can compromise healing. Overall, fasciectomies offer the benefit of removing more diseased fascia but are accompanied by increased morbidity related to a more extensive exposure.

8.4. Post-operative protocol

Most surgeons utilize some form of rehabilitation to prevent further contractures and maintain ROM. After surgery, patients are typically immobilized in an extension-based splint for 2–3 days. Wounds are closely monitored following surgery to ensure adequate healing and to identify any barriers to healing such as infection, hematoma, or skin necrosis. Patients are often referred to a hand therapist within the first week of surgery for wound care, scar management, range of motion exercises, and splinting techniques to prevent contracture formation. A variety of splinting protocols and types of orthoses have been described. In general, protocols may involve static or dynamic splinting and be utilized at different periods of the day. Despite the widespread use of post-operative splinting, studies have found no strong evidence they are effecting in preventing loss of extension or recurrence. Collis et al. randomized patients to night

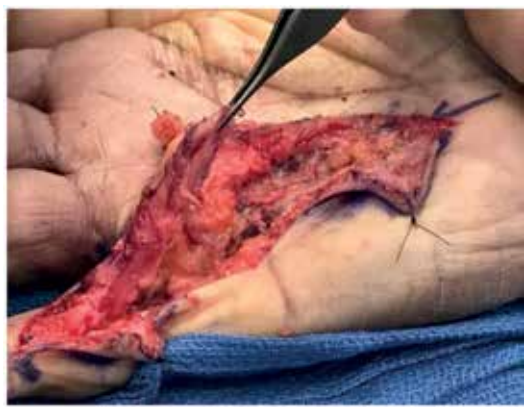

Figure 5. An intraoperative image of a patient undergoing a limited fasciectomy for a Dupuytren's contracture of the small finger. A Brunner's incision was utilized to exposed the disease tissue. The Dupuytren's cord is being elevated by the forceps.

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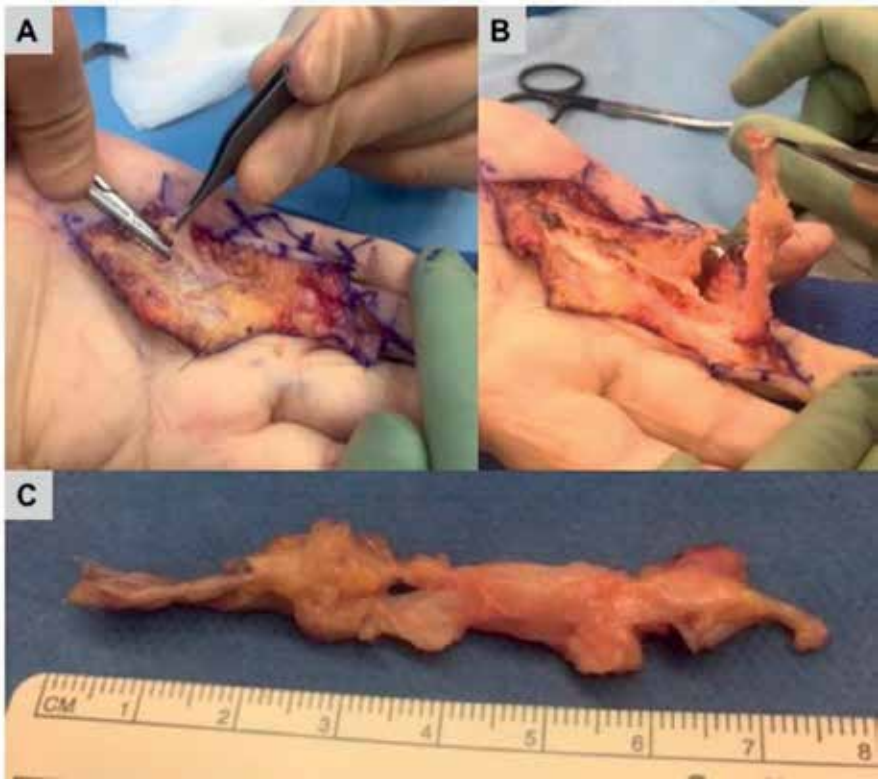

Figure 6. An intraoperative image of a patient undergoing a limited fasciectomy for a Dupuytren's contracture of the ring finger. (A) A Brunner's incision was utilized to exposed the disease tissue. The longitudinal band of the palmar aponeurosis is being elevated. (B) The diseased fascia was meticulously elevated from proximal to distal. (C) The diseased fascia after removal from the digit.

time extension orthoses and hand therapy or hand therapy alone and reported no differences between the two groups in terms of active ROM or hand function [72]. A similar study found night time splinting offered no benefit in terms of ROM and function at 1 year after surgery and recommended splinting should only be utilized when extension deficits occur [73]. Overall, less invasive procedures allow for earlier rehabilitation and a shorter recovery period. In addition, wound healing often dictates how fast a patient can progress following surgery. A segmental aponeuroectomy may involve a 2–3 week recovery period whereas a fasciectomy may involve a much longer recovery period.

9. Patient outcomes

Overall, surgery is an effective method of treating Dupuytren's contractures and improving patients' hand function. In a survey of over 1100 patients who underwent surgical treatment 75% reported almost full or full correction of their contracture [74]. Zyluk et al. reported patients had significantly improved hand function as measured by the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire after undergoing a subtotal fasciectomy [75]. In

addition, patient's experienced a significant improvement in the total loss of extension from an average of 80 to 10°. Another study prospectively evaluated 90 patients undergoing a fasciectomy for a 60-degree or more deficit in total active extension and reported 81% patient satisfaction with function, 87% reaching functional ROM, and significantly improved DASH scores at 1 year follow up [76].

Further analysis of surgical treatment has lead authors to identify certain characteristic associated with better outcomes. Patients with MCP contractures are more likely to achieve full intra-operative correction (Donaldson). However, correction of PIP contractures has a stronger correlation to improved hand function when compared with correction of the MCP joint [77]. Surgical treatment has focused on PIP contractures to maximize intra-operative correction and improve functional outcomes. Surgeons have tried releasing the PIP capsule with a fasciectomy to improve PIP correction, but there is no strong data to support whether it is effective [78]. Zyluk et al. reported younger patients had a significantly greater functional improvement after surgery as measured by the DASH score [75]. Studies have also cited the extent of preoperative deformity, incomplete correction, and multiple involved digits as factors affecting post-operative functional outcomes [75, 78–80].

Despite multiple studies reporting good outcomes after surgery, there are limited randomized studies comparing outcomes after different operative techniques. Ullah et al. found no difference in ROM or recurrence in 79 patients randomized to either direct closure with z-plasty or firebreak skin grafting after a fasciectomy, however, patients with skin grafting had an increased incidence of hypoesthesia [81]. Van Rijssen et al. randomized patients to percutaneous needle fasciotomy and limited fasciectomy and reported the PNF group was significantly higher recurrence rate (76.8 vs. 20.9%, $p < 0.001$) and lower VAS satisfaction scores (6.2 vs. 8.3, $p < 0.001$) [62]. Further prospective, randomized studies reporting functional outcomes, complications, and recurrence rates are necessary to recommend any surgical procedure.

10. Recurrence

Recurrence of a Dupuytren's contracture is a common event even after successful initial treatment. A systematic review analyzed 51 studies and reported recurrence rates ranged from 0 to 71% [82]. Furthermore, recurrence rates are difficult to assess as there is considerable variation in the criteria used to define recurrence. Some authors report the presence of any diseased tissue after treatment while others include only contractures necessitating re-operation. More recently, studies have tended to define recurrence as a 20–30° loss of extension in a successfully treated digit. A recent randomized study defined recurrence as a 20° reduction of total passive extension in a successfully treated digit and reported a 20.9% 5-year recurrence rate after limited fasciectomy [62].

Multiple studies have focused on identifying factors which may predispose patients to recurrence. The dramatic variability of recurrence rates may be due to the heterogeneity of the presentation of Dupuytren's itself, as many patients may have more aggressive biology associated with "Dupuytren's diathesis", whereas others may have more mild disease. Dupuytren

diathesis is a term coined by Hueston describing certain characteristics related to severe disease and increased recurrence [83]. Hindocha modified the criteria to include the following features within a Northern European population: male sex, <50 years old, bilateral disease, affected parent or sibling, and presence of Garrod's nodes and reported patients with all 5 features had a recurrence rate of 71% [84]. However, other studies have failed to demonstrate a significant correlation with recurrence among all 5 diathesis criteria. Van Rijssen reported only older age was found to delay recurrence after PNF and limited fasciectomy [62]. PIP joint contractures have an elevated recurrence rate after surgery compared to the MCP joint. Donaldson et al. reported 34.2% of fully corrected PIP joints experienced at least some loss of correction compared with 12.2% of MCP joints [80]. Patients with severe preoperative PIP contractures greater than 60°, incomplete correction, and poor post-operative compliance had significantly worse recurrence [78].

Surgical management of recurrent disease is challenging as anatomic landmarks and tissue plans become difficult to distinguish. There is currently limited data regarding the preferred surgical treatment. Roush and Stern compared dermatofasciectomy with a skin graft, interphalangeal joint arthrodesis, and fasciectomy with local flaps among 19 patients with recurrence after a prior surgery [85]. The fasciectomy cohort was the only group to significantly maintain total active motion at final follow-up, but all three groups had similar patient reported outcomes. It is important patients are aware of the risk of recurrence prior to their initial surgery and personal factors which may increase their likelihood of recurrence.

11. Complications

Despite good outcomes after surgical treatment of Dupuytren's contractures surgery is not without complications. A 20 year systematic review of complications by Denkler et al. reported an average major complication risk of 15% with complication rates ranging from 3.6 to 39.1% [86]. Specific complications after limited fasciectomy included the following: wound healing problems, 22.9%; flare reaction, 9.9%; complex region pain syndrome, 5.5%; nerve injury, 3.4%; infection, 2.4%; hematoma, 2.1%; and digit artery injury, 2%. Severe complications include tendon rupture or loss of the digit but are extremely rare. Patients with severe flexion contractures are more at risk of experiencing a complication [87]. Smoking and diabetes, however, has not been identified as an increased risk factor for wound healing problems after surgery [88]. Patients undergoing revision surgery for recurrence are most at risk for complications, especially neurovascular injuries due to scar tissue and loss of anatomic landmarks. Neurovascular injuries have been reported as high as 10 times more common in revision surgeries for recurrence [86].

12. Conclusion

Dupuytren's disease is a unique fibroproliferative disorder of palmar fascia likely resulting from a complex interplay of genetic and environmental factors. Despite extensive research,

its etiology remains unclear and treatment methods remain palliative. Observation is an acceptable form of treatment for mild disease and patients with low functional status. Collagenase injections and percutaneous fasciectomy are becoming more common as an initial treatment method for patients with isolated disease and a palpable cord. Surgical indications typically include a flexion contracture of >30° at the MCP joint and 10–15° at the PIP joint but are also influenced by patients' functional goals. Limited fasciectomy is the preferred method of surgical treatment by most surgeons. However, there is insufficient high quality evidence comparing different methods of treatment. In general, starting with a less invasive treatment is a reasonable approach; the recurrence rate may be higher, but patients will benefit from a quicker rehabilitation and a lower complication rate. Limited fasciectomy can then be reserved for more severe disease, initial treatment failures, or recurrence. PIP contractures, however, may benefit from earlier intervention due to their impact on function, increased likelihood of incomplete correction, and higher recurrence rate. Overall, surgery is an effective treatment for Dupuytren's contractures but complications can occur especially related to wound healing, and many patients experience recurrence. Further research is still needed to compare treatment modalities and determine appropriate indications.

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Chapter 6

Crush Injuries of the Hand Part I: History, Mechanism and Pathomechanics

Roohi Sharifah Ahmad and Pho Robert Wan Heng

Additional information is available at the end of the chapter

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Abstract

This chapter, the first of two; deals with the basics of crush injuries of the hand as opposed to crush syndrome. The definition is explained and the differences are outlined. A journey through the historical review of the causative mechanisms gives the reader an insight into the machines inflicting a spectrum of injuries and their sequelae. We see how the tools either remain the same or change with time, exacting similar types of injuries but in a different manner and timescale due to mechanisation. Thus it is vital to grasp the mechanics to get a basic understanding of the pathomechanics, enabling one to address the injury by reversing the inflictive force while maintaining respect for the machine. These are humbling injuries that require experience, expertise and enterprise by a dedicated and cohesive team always open to learning.

Keywords: crush, hand, injuries, mangled extremity, trauma

1. Introduction

Crush injuries of the hand pose a challenge to even the most accomplished of hand surgeons, whether it is a minor fingertip injury sustained by getting squashed in a closing door or a high pressure compression injury involving the palm or wrist.

A **crush injury** is defined as compression of the extremities causing muscular and neurological disturbance [1] and in the upper limb is sustained when the fingers, hand or wrist are caught between two surfaces (sharp, blunt, smooth or irregular) forcibly producing damage to the skin and its enclosed contents of soft tissues and bone. The degree of damage is proportional to the amount of force applied per square inch and the duration the compression is in place.

Thus a crushing element is present in almost all hand injuries be it distributed over a narrow segment as in a guillotine amputation of a finger or diffusely spread as in a roller injury.

Prolonged compression in heavy machinery in a more proximal part of the limb may induce **additional** systemic sequelae known as the **crush syndrome**. This was first described in the German language literature by Von Colmers, following the 1909 Messina earthquake and by Frankenthal during the 1916 World War I air raids as cited in Better [2]. The English Language literature, however, was only enlightened by Bywaters and Beall after the 1940 London 'Blitzkrieg' of World War II, where they outlined the pathogenesis of crush syndrome and its potential systemic effects of myoglobinuria leading to acute renal failure causing the patient's demise hours or even days later [3]. He described alkalinisation of urine as a method to prevent the acute renal failure and subsequent deterioration, which has stood the test of time. Michaelson defined continuous prolonged pressure on the limbs of at least 4 hours duration prior to extrication as causing crush syndrome [4]. Fortunately, crush injuries to the hand distal to the wrist have less systemic manifestations primarily due to a smaller muscle bulk. They are however otherwise no less dramatic and have evolved over the years associated with technological developments in human endeavours (**Table 1**).

Time and causative factor	Authors	Year
Agricultural age		
Machines on the farm		
Corn picker injury	Robinson and Hardin	1955
	Campbell DC et al.	1979
	Gorsche and Wood	1988
Grain auger injury	Grogono	1973
	Beatty et al.	1982
Farm machinery	Simpson	1984
Farm-related Injuries	Chun	1999
Woodwork related mishaps		
Woodworking tools	Heycock	1966
Chain saw injuries	Haynes et al.	1980
Wood splitter injuries	Jaxheimer et al.	1981
Industrial age		
Industrial machines		
Wool carder	Smith and Asturias	1968
Wringer injury	MacCollum et al.	1952
Electrical machines		
Escalators	Campbell Reid	1973

Time and causative factor	Authors	Year
Meat mincers	Al-Arabi and Sabet	1984
Sugarcane juice extracting machine	Rajput and Daver	1999
Dough sheeter	Carriquiry and Arganaraz	2005
Noodle-making machine	Ju et al.	2015
Motors		
Rotary lawn mowers and snowblowers	Barry and Linton	1977
Roll over injury	Harris and Wood	1978
Roll bar hand	Charters and Davis	1978
Overturning motor vehicle	Mehrotra and Crabb	1979
Forklift		1999
Information age		
Roping injury	Morgan	1984
Roping injury	Kirwan and Scott	1988
Pay phone receiver cord	Lesavoy	1984
Sports related		
Soccer	Curtin and Kay	1976
Karate	Nieman and Swann	1977

Table 1. Causes of crush injuries over the last 50 years.

2. Historical review

2.1. Mechanism of injury

2.1.1. The agricultural era

This period brought about some of the most devastating effects from the corn picker injury as first described by Robinson in 1955 and later by Campbell in 1979 and then Gorsche in 1988 [5–7], and later by the equally if not more devastating Grain Auger injury which cut swathes at multiple levels [8, 9]. In the mid 1960s to early 1970s when the oil embargo of the Arab states diverted energy sources away from fuel to firewood, a higher incidence of injuries with wood working tools was reported by Heycock in 1966 [10], the modern (and fatalistic) version of which is the chainsaw or circular saw injury (**Figure 1**) [11] and the motorised wood splitter injury [12]. The advent of industrialisation into the agricultural sector saw farm machinery and farm-related injuries coming into the scene in the 1980s and 1990s being a challenge due to the high contamination [13, 14].

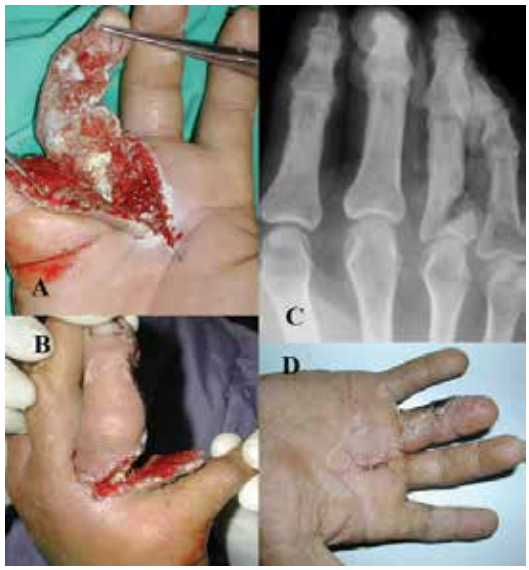

Figure 1. A, B: A circular chainsaw used to cut meat went through this gentleman's 3rd web space damaging flexor tendons, nerve and pulleys, plus **C:** Creating a gap in the proximal phalanx corresponding to the gauge of the saw. **D:** Although secondary infection complicated the healing, the flap survived and function was restored.

2.1.2. The industrial age

The advent of **the industrial age** saw its own share of mutilation with workers spinning wool on an electrical wool carder [15] sustaining a unique injury thereof with the fingers undergoing a crush and the spikes inflicting a horrific-looking but benign injury (**Figure 2**).

Children were the victims in 80% of "The Wringer Injury" as cited by MacCollum was first described in 1938 [17]. It wreaked havoc for 45 years till production was stopped in 1983 via legislation [18]. In the late 1960s and early 1970s, there was a flood of children getting their hands caught in escalators and meat mincers each presenting with their unique brand of mechanism and challenge [19, 20]. The former primarily caused a deep avulsion or a degloving (**Figure 3**), while the latter had two levels of injury: a multilevel cutting injury first like a miniature auger (**Figure 4**) and a mincing mechanism sustained later.

Peculiar but not exclusive to South and South East Asia is the sugarcane juice extraction machine which was described in 1999 [21] and has also evolved from manual to electric. This produced devastating injuries similar to the dough sheeter injury [22] with components of grinding, compression and avulsion, making it difficult to salvage digits [23]. In our experience, most end up with a metacarpal hand for the machine is very unforgiving and the infection due to bacterial contamination challenging to eradicate, requiring meticulous, repeated débridements in the primary setting (**Figure 5**). Another unique Asian injury is the noodle machine, which causes a degloving injury (**Figure 6**), of which in the English literature only one article mentions it in passing [24]. A more widespread but less reported one is the coconut grinding machine which causes a similar injury to the grain auger (and meat mincer) but of a smaller scale (**Figure 7**).

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Figure 2. A, B: Images taken form Strauch filter blog [16] depicting how the wool is fed and what the machine looks like. **C:** The clinical picture where the digits (mostly middle and proximal phalanges) are crushed. **D:** Proximally the spikes, depending on their circumference and length inflict minimal harm. **E:** There was no neurological deficit in this gentleman and the flexors were fine as can be seen.

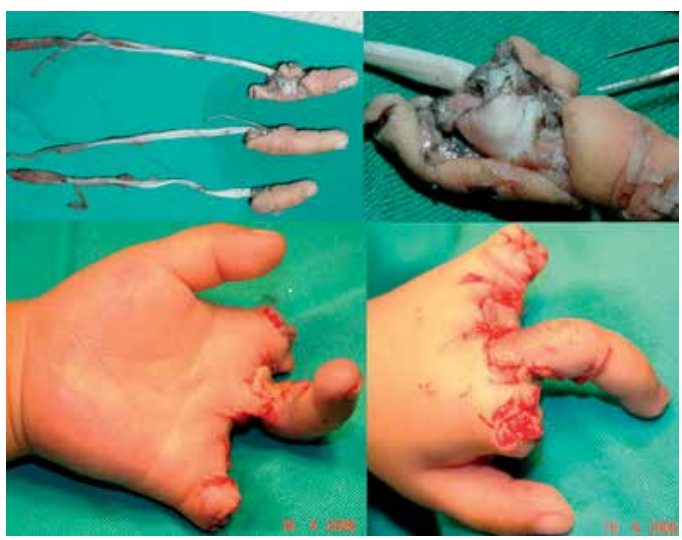

Figure 3. Anticlockwise from top left: A 3 year old girl caught her left hand in an escalator when she fell and had an avulsion of all her fingers. Only the middle pinked up after replantation. There was severe torsion and avulsion as shown.

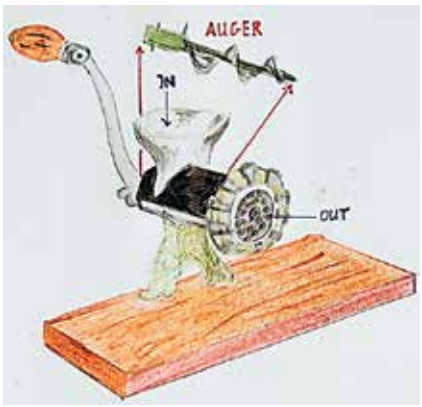

Figure 4. The manual meat mincer is not as damaging as the electrical one. The meat is placed into the funnel, pushed in and drawn into the mini auger mechanism (drawn outside of the black tube) which then forces it out through the small perforations (2–3 mm in diameter). If a hand is caught, it will be cut in swathes and squeezed out through the exiting mechanism; beyond repair or reconstruction. Usually the machine has to be brought to hospital and the patient placed under anaesthesia before extrication.

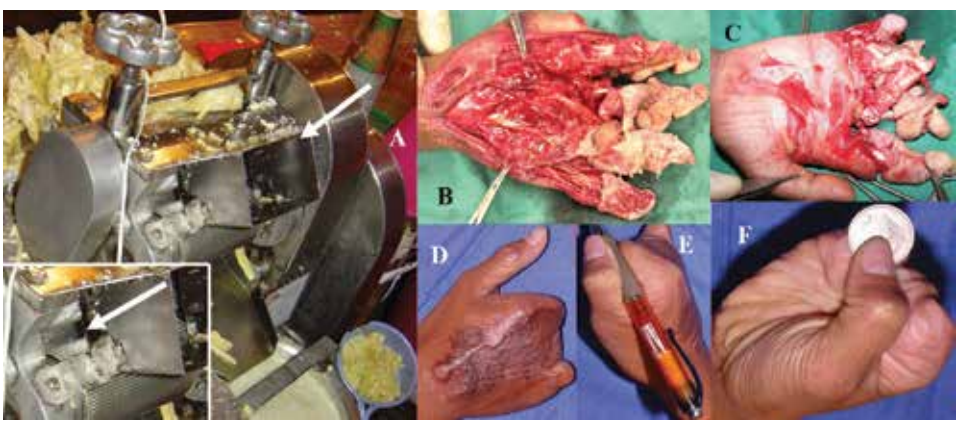

Figure 5. A: The sugarcane juice extraction machine feeding box (right white arrow) is where the cane is pushed into. It squeezes the cane between two rollers (left white arrow, inset) with an adjustable width in between (8–15 mm). **B, C:** Besides causing severe crushing (distal to proximal) and mangling the extremity, there is also extensive avulsion since the victim usually is trying to pull out the hand in the opposite direction to the rollers. **D-F:** This was the only patient in a series of six cases over a 2-year period [23] whose thumb was left intact. Usually only the metacarpals were spared and saving the remaining was an arduous if not impossible task.

In the **vehicle** category is the powered industrial vehicle (PIV) and although the literature describes how 70% of PIV injuries are attributable to forklifts [25], there are no specific articles on hand injuries caused by forklifts [26]. This is we feel a special entity which needs to be dealt with because it causes extensive damage to the skeleton as well as the soft tissues and needs rigid and rapid fixation best achieved with a simple form of mini external fixator (**Figure 8**). Spring and winter brought their own brand of injuries in rotary lawn mowers and snow blowers [28], while the motor vehicle left its own special mark, described as the roll

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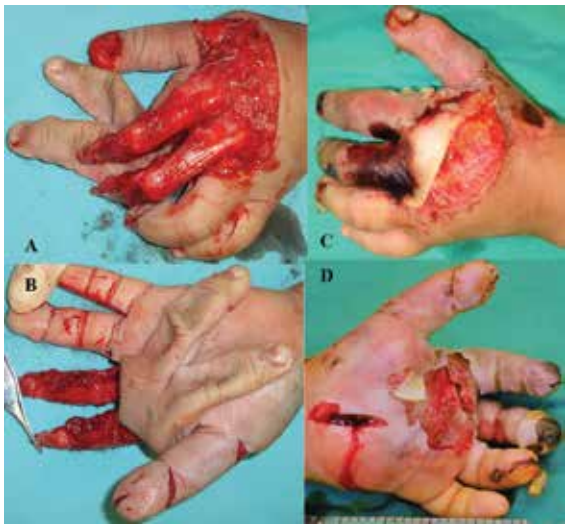

Figure 6. A, B: A 5 year old boy had his left hand caught in the escalator and the radial side was completely degloved. The veins were also thrombosed but we could anastomose one vessel on the thumb. **C, D:** As can be seen, the thumb and index finger were still pink a week later but part of the middle finger skin (only) had necrosed.

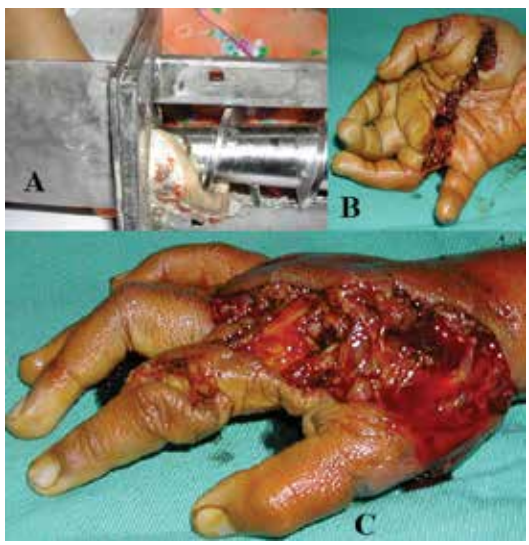

Figure 7. A: Coconut grating machine. A smaller version of the grain auger but just as devastating. Usually these devices need to be removed with the help of the fire department and sometimes under sedation or anaesthesia. **B, C:** Extensive crushing of the bone and soft tissues is seen at two levels.

over injury or roll bar hand in 1978 by Harris and Charters, respectively [29, 30] and subsequently by Mehrotra and Crabb as hand injuries sustained in the overturning motor vehicle [31]. Typically, the victim would have the hand outside the window during the accident or it is grazed along the gravel – what we call the "brake pad injury" (**Figure 9**).

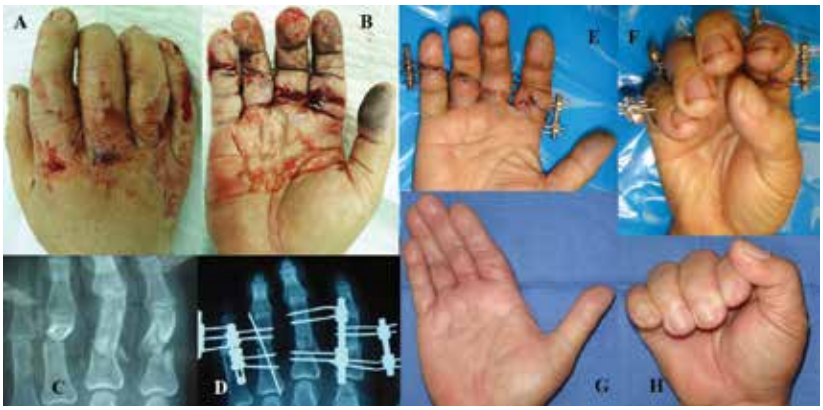

Figure 8. A, B: This gentleman was a supervisor who inadvertently got his right (dominant) hand caught between the ceiling beam and the forklift prongs. **C:** A clear line of injury (fractures) is seen across the proximal and middle phalanges. He had a heart condition and we were given 2 hours time to fix all. **D-F:** Mini external fixators from Waldemaar Link® [27] made a rapid, rigid fixation possible and a mobile hand is seen within 5 days. **G, H:** 3 months post-operatively, he has a good range of motion and is able to make a grip.

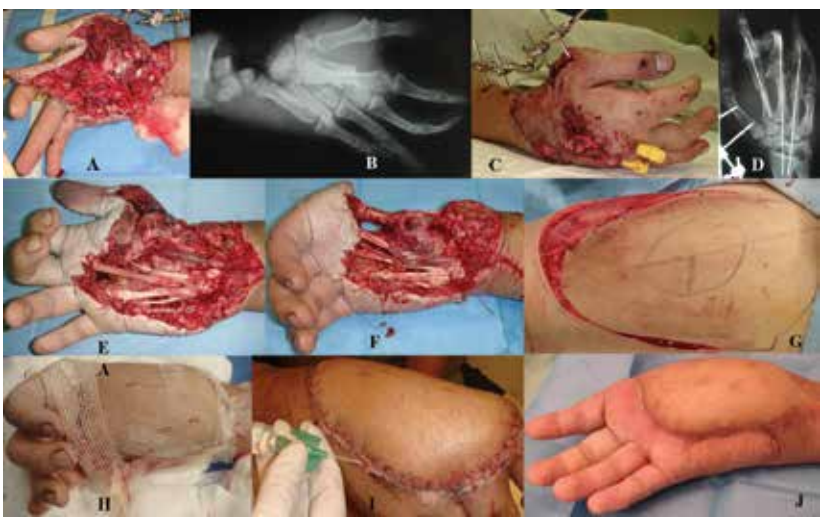

Figure 9. A, B: A 30 year old Army captain had a roll over injury in his car and hit a metal divider which all but severed his right hand at the radio-carpal level. He presented from another hospital 20 hours later. Salvage was attempted because it was his right hand. **C, D:** Revascularisation, debridement and tendon repair performed at 24 hours post-trauma and held with Kirschner wires and an external fixator. **E:** Infection destroyed the revascularisation and 3 days later the thenar muscles became necrotic. **F:** However, the thumb ray was only amputated after a delay in consent of 5 days. **G, H:** A free lateral thigh flap was performed to cover the defect and **I:** The battle with infection was finally won a year later. **J:** He felt "functional" although he had lost his thumb and did not want a pollicisation.

2.1.3. The information age

In this era, electrical equipment and wiring had peculiar stringing injuries as described by Morgan in 1984 [32] and as roping by Kirwan and Scott in 1988 [33]. Pay phone receiver cords [34] and power drill cords also cause similar injuries where the cord wraps around the hand or forearm like a vise cutting off blood supply and causing ischaemia to the limb (**Figure 10**).

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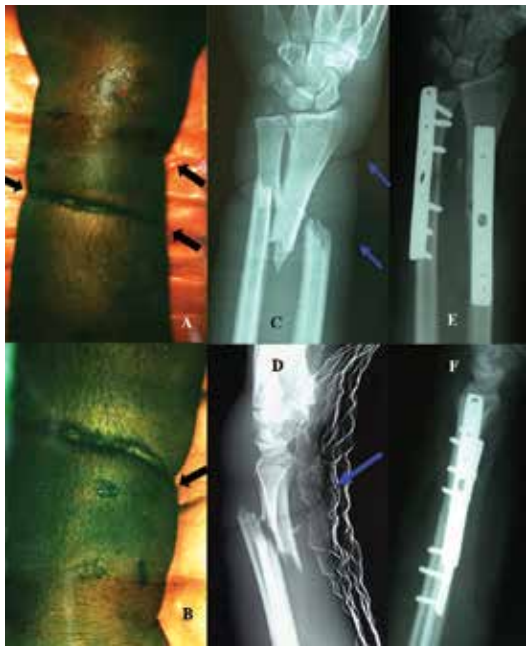

Figure 10. The deep indentation caused by the electrical cord (black arrows in **A & B**) can be seen with the corresponding marks on the radiographs (blue arrows in **C & D**), along with the bony injury. In this patient, prior to open reduction and internal fixation, the vessels were explored and found to be intact but in spasm. The vessel spasm was overcome by 10% Xylocard and warm saline, and subsequently bony fixation was carried out, **(E, F)**.

2.1.4. Sport

A number of different types of hand injuries have been described while playing soccer but these are mostly fractures or associated ligamentous injuries with less of a crushing element to the tissues [35]. Karate is a sport, which may cause a crush injury to the hand or forearm since the hand is used as a weapon to demolish bricks and other hardware [36].

Thus we can see the spectrum of change in the pattern of crushing injuries to the hand but though the cause may be different, the mechanism and resultant effects still pose a challenge to the modern-day hand surgeon.

2.2. Pathomechanics

The pathomechanics of a crush injury will vary according to the manner in which the injury was sustained. The damage done is related to the **force** of the injury **(both** in **magnitude** and **direction)**, the **velocity** of the impact and the **surface area** of the crushing. The damage sustained is also dependant on the **site** of injury, the surrounding skin and its contents.

Therefore, the **zone of injury** sustained is a function of the applied **force**, the **velocity** and the **width** of the offending object. These three main factors will determine the outcome and extent of the injury. The **duration** of compression as well as compounding factors such as **friction**, **heat**, **cold**, **chemicals** and **contamination** add further damage to the injured area.

2.2.1. Magnitude of force and velocity

The force may be a **low-energy** mechanism with resultant closed injury and fewer stigmata of damage. Typically, this may be due to a door or a drawer closing on the fingertip producing a subungual haematoma, a nailbed injury, a mallet deformity or a tuft fracture of the distal phalanx (**Figure 11**). The milder ones may not even present at the casualty department but may be seen later in the clinic. A more severe form seen especially in children called a Seymour Fracture occurs where the nail plate is avulsed proximally with an associated nailbed injury and a distal phalanx (P3) fracture (Salter Harris I) through the epiphysis (**Figure 12**). The middle finger being the longest is most commonly involved. Soft tissue interposition and instability dictate operative repair and fixation otherwise complications may include infection, nail disturbances, growth arrest and deformity [37].

A **high energy** force with a high velocity of impact would cause serious damage not only at site of impact but can cause degloving of the proximal part as well, from the dynamic force of the hand pulling away. These injuries are sustained in a road traffic accident or high pressure punch presses such as printing presses (**Figure 13**).

2.2.2. Direction of force

The crushing mechanism may be **uniplanar** as in a heavy object falling from a height onto the hand or **multiplanar** with either an associated *torsional* or a *tractional* force. Grain augers exert a *torsional* force and are used for raising grain and they cause characteristic multiple swathes equidistant to the spiral turns of the auger blade drawing the limb in with each turn (**Figure 14**). A similar mechanism is used in the meat mincer and the coconut grating machine (**Figures 4**, **7**). An example of a *tractional* force can be seen in Roller belt injuries (**Figure 15**) as well as those due to the sugarcane juice extracting machine where severe avulsion or degloving of tissues can occur due to the pulling action, along with devastating superinfection due to microbial (*Pseudomonas Ae*) contamination [23].

2.2.3. Width of crushing force

The width of the instrument of injury determines the depth and extent of bone and soft tissue damage.

Figure 11. A: Typical fingertip injury in a child due to entrapment in a drawer or a door jamb. Note the proximal nail plate extrusion. **B:** Simple reduction of the nail plate under GA is required with **C, D:** Suturing of the pulp with a Vicryl® 7/0 spatulated needle.

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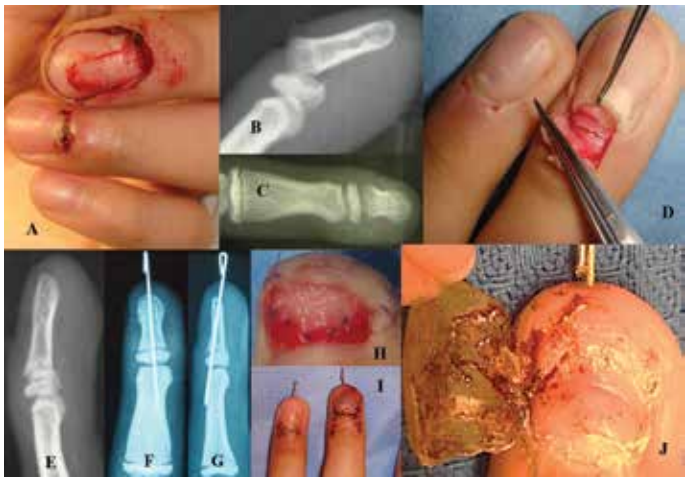

Figure 12. A: Typical Seymour fracture with haematoma and nail plate avulsion from base and flexion deformity of the middle finger. This deformity is due to the metaphyseal attachment of the flexor to the distal phalanx (P3), while the extensor attachment is more proximal at the epiphysis. **B, C:** X-ray shows a fracture through the epiphyseal plate but it may be missed in the AP view. **D:** The nail plate is elevated and two parallel skin incisions lifting the eponychium reveal the nailbed fracture in the germinal matrix. **E:** The fracture is reduced, then **F, G:** Pinned. **H:** We use spatulated 7/0 Vicryl® to suture the nail bed**. I:** The nail plate is reinserted under the eponychial fold after repair. **J:** After 2–3 weeks the nail plate can be removed to allow the new nail to grow out.

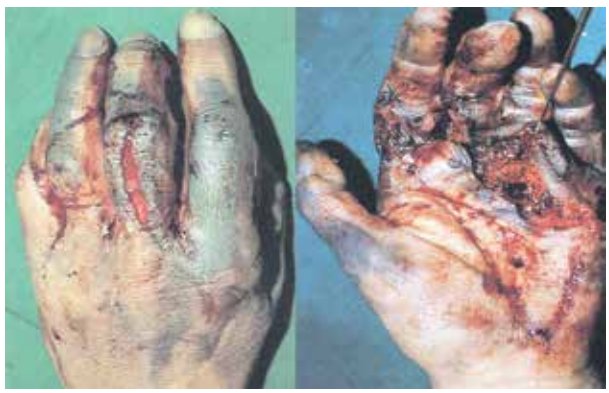

Figure 13. Left hand caught in a printing press. The ink marks can be seen staining the hand. To allow accurate assessment of circulation, these have to be scrubbed off. There is a longitudinal burst laceration seen over the dorsum of the middle finger over the proximal phalanx as well as a degloving injury in the palmar area (skin hook).

Pressure = Force per unit area.

Therefore, for the same force, the smaller the unit area, the higher the pressure concentrated over that minute area. Thus a sharp instrument with a lesser amount of force (sharp knife = amputation) might slice through tissue whereas a compression over a wider area (hammer = burst laceration) would cause more of a crushing injury.

The direction of the force is also important, whether it is along the tissue planes or perpendicular to it. Thus a vessel may be sliced in a transverse fashion – in case of a complete transection,

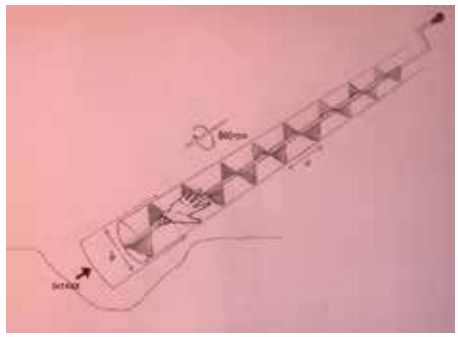

Figure 14. The grain auger mechanism uses the Archimedes principle to churn up water or grain against gravity. The spiral blades are approximately 6 inches apart (w) and the diameter (d) of the tube is also 6 inches. Thus the pitch is 1–1. It runs at 800–900 revolutions per minute and therefore moves grain up at a speed of 400 feet a minute or 7 feet a second!.

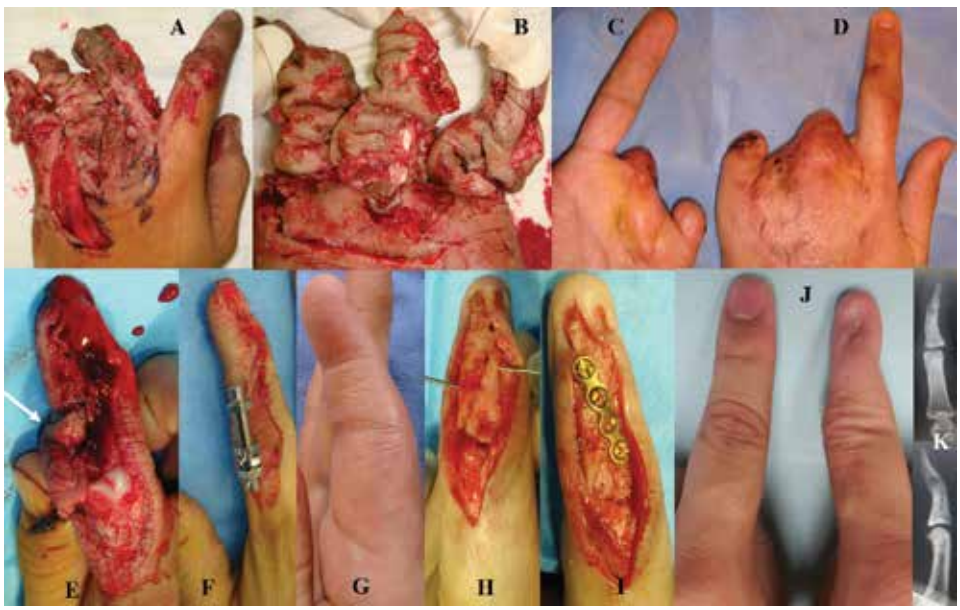

Figure 15. Top row: **A, B:** Shows the hand of an engineer that got caught in a running conveyor belt that was faulty. Note the 'rolled' skin folds and how the flexor tendons remain intact. **C, D:** All but his index and thumb were mangled which afforded him a pincer grip. Bottom row: **E:** A 32 year old mechanical engineer also got his finger caught in a roller belt which grazed the radial half of his ® dominant index finger, leaving only a sliver of rolled skin (white arrow) which **F:** Was used as a skin graft in the repair and skeletal stabilisation. **G:** He only had 20° of flexion after removal of the external fixator at 6 weeks. **H, I:** Reconstruction was performed using a bone graft and plating, 4 months post-trauma and physiotherapy. **J:** Size is smaller but he has full power and two-point discrimination of 4 mm. **K:** The radiographs show good fusion of the DIPJ and a clear PIPJ. Final ROM was 10–75°.

it will go into spasm and contract, but a partial one may be held open and still bleed – or scathed along its length (**Figure 16**). The latter will usually present with hypovolaemic shock due to profuse uncontrollable bleeding. Pressure dressing should be applied and exploration performed in the operating room.

2.2.4. Site of injury

The dorsum of the hand has minimal soft tissue padding and is relatively vulnerable to **bony injury**. A metacarpal fracture commonly occurs with a direct blow on the dorsum of the hand. Bony injury often results from the high energy impact of blunt objects related to the factors of force, velocity and width of the striking objects and may present in a spectrum of varying depths of involvement and comminution (**Figure 17**), from periosteal stripping, to unicortical fracture, to transverse, oblique, spiral or comminuted fracture, to segmental fracture and segmental bone loss.

Soft tissues over the palmar aspect are fairly well protected by the thick and highly sensitive glabrous skin. Neurovascular bundle and flexor tendon injuries are seldom seen in closed injuries. In open fracture of the phalanges, associated neurovascular and flexor tendon injuries must be looked for. The flexor tendons are very resistant to injury and usually are the last to be severed (**Figures 5** and **15B**). In the mid-palmar space, which is a relatively confined region holding soft tissues contained by a thick fascia, a sudden compression with high pressure may lead to bursting injuries, resulting in rupture and extrusion of muscles (**Figures 18**, **19**).

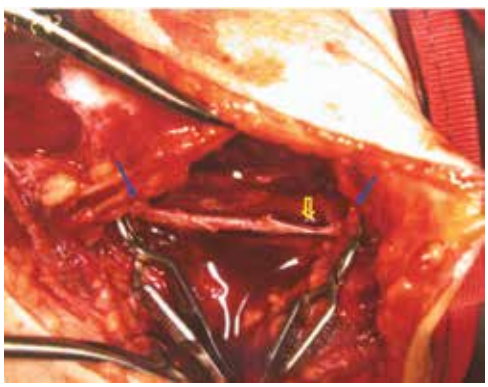

Figure 16. Stab injury to the right arm of a young gentleman who was hemodynamically unstable at presentation with a compromised circulation to his right upper limb. The brachial artery had been severed completely (held in bulldog clamps – solid arrows) and was in spasm but the vein was scalloped (hollow arrow) by the sharp instrument used in this assault and the culprit in the profuse bleeding. Picture courtesy of Professor Lim Beng Hai, Dept of HRM, NUH.

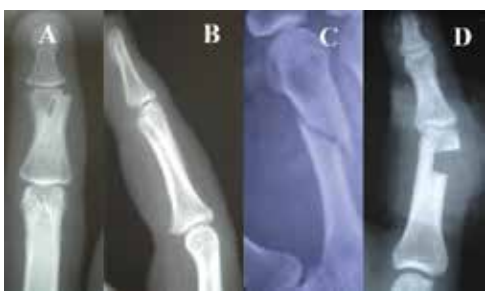

Figure 17. Varying depths of bony involvement: Radiological features **A, B:** Unicortical fracture which was initially missed in the A&E department, **C**: Short spiral fracture of 2nd metacarpal, and **D:** Segmental bone loss in the proximal phalanx the width of which corresponds to the thickness of the grinding axe.

2.2.5. Other factors

Thermal necrosis may be present due to friction, as in roller injuries or heat, chemical and electrical burns which cause either partial or full thickness skin loss or due to hot compression presses which result in full thickness loss even up to the deep muscle layer of the hand (**Figure 20**).

The roller injury is special in that it commonly results in distally based avulsion flaps and may be associated with friction burns [38]. The size of the gap between the rollers as well as

Figure 18. A, B: This lady's dominant right hand was caught in between two compressive surfaces and sustained burst lacerations to index, middle and ring fingers but the flexor tendons can be seen intact. The pulleys however, had to be reconstructed. **C, D:** The severity of comminution can be appreciated in the radiographs. **E, F:** The index finger had good refill and was fixed with cross k-wires: **G.** The middle finger could not be salvaged due to severe damage to both digital vessels. The periosteal sleeve of the proximal phalanx was shredded and also undertaking reconstruction would have retarded rehabilitation of the hand as a whole. **H, I:** The ring finger had multiple comminuted fractures and had to be revascularised. She went on to heal with some stiffness, but had pinch and grip functions.

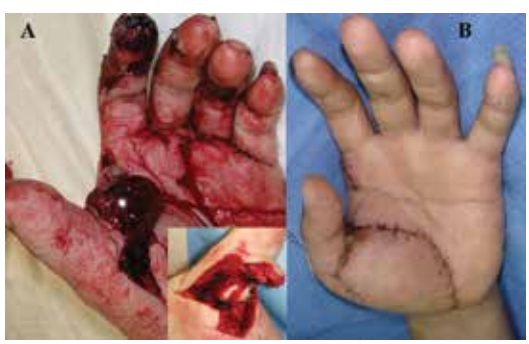

Figure 19. A: A 30 year old caught his left non dominant hand in a 200Kg compression machine while he himself was operating the lever. Debridement could only be done 6 hours later. **Inset:** After removal of the haematoma, it was noticed that the muscles were ischaemic, the pulleys ruptured and the MCP joint volarly dislocated. **B:** The joint was reduced and held with a K-wire. The thenar muscles that were necrotic had to be excised and carpal tunnel was released in anticipation of the oedema. He regained excellent motion but had reduced strength as expected.

the padding is important to determine the amount and force of crushing. If there is an automatic stop mechanism, there is some tendency to minimise damage. An examiner should not be foxed by superficial friction burns. A **degloving injury** of skin and deep tissues may not be apparent especially in a closed injury or incomplete avulsion. The resultant shearing off of blood vessels in the subcutaneous plane may lead to secondary thrombosis of the blood vessels resulting in fat necrosis and delayed necrosis of overlying skin [39]. A direct injury to the muscle or a compromised circulation of the forearm and hand may eventually lead to Volkmann's ischaemic contracture, an early symptom of which is pain on extension of the digits (**Figure 21**). A narrow gap in the rollers would inevitably involve severe crushing of muscles, nerves and the skeletal framework. For the novice, a simple guide to the extent of crushing may be evaluated by the degree of bony comminution in the X-ray (**Figure 22**).

Figure 20. A, B: The ring and small fingers of this man's right hand were caught in a cardboard box-manufacturing machine of an ice-cream factory. He sustained full thickness burns of all structures down to bone. Revision amputation had to be carried out.

Figure 21. A 22 year old man sustained a roller crush injury of his left arm up to mid-humerus. He developed Volkmann's ischaemic contracture with atrophic non-union and shortening of his distal radius fracture, resulting in radial deviation. He also had patchy involvement of both median and ulnar nerves typical of ischaemic aetiology. **X-ray:** An ulnar shortening osteotomy and radial bone grafting was done to correct the deformity along with extensor adhesiolysis and median and ulnar nerve neurolysis.

2.3. Pathogenesis of crush syndrome

Compression of muscular segments of the limb is the basic mechanism underlying the pathogenesis of crush syndrome. The relative contribution of compression leading to ischaemia of the muscles and direct injury to the muscle leading to necrosis is difficult to separate. Mechanical compression alone with an adequate vascular supply (warm periphery with palpable pulses) has been shown to cause significant pathological changes in skeletal muscle by as early as 60 minutes as shown by Better and Stein and cited in Burzstein and Carlson [40]. This compares unfavourably to a warm ischaemic time of 6 hours – without compression [41], leading Burzstein and Carlson to conclude that *skeletal muscle is more sensitive to mechanical compression than ischaemia*. This may be due to the fact that in compression intramuscular pressures may reach as high as 240 mmHg [42] which is thought to compromise the microvasculature of skeletal muscle. This may cause alteration in myocyte function in terms of calcium flux across mitochondrial and plasma membranes.

In ischaemic injury however, reperfusion results in further damage to skeletal muscle resulting in the coinage of the term "reperfusion injury". The offending substances are O_2-derived free radicals such as super oxide, H_2O_2 and hydroxyl ions, which cause parenchymal and microvascular endothelial damage especially with reperfusion. Compounds such as super oxide dismutase (SOD) and catalase which when administered, inhibit or neutralise these radicals, have been shown to limit the reperfusion injury in the affected tissue and its microvasculature [43, 44].

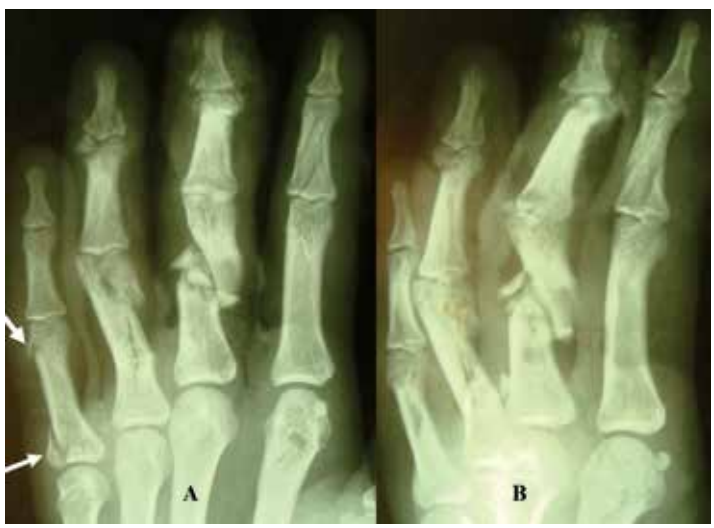

Figure 22. A, B: Compare these AP and lateral radiographs to the clinical picture of the patient in **Figure 2**. The degree of comminution (and displacement) is worst in the proximal phalanx of the middle finger followed by the ring and small. The former two would best be fixed internally with mini plates. The small finger may get away with a lag screw fixation – the abductor digiti minimi inserts at the base while the collateral ligament attaches at the neck. The middle phalanx of the index finger also has significant comminution, but no displacement and would do well with a mini external fixator. Note the punched hole in the 2nd metacarpal.

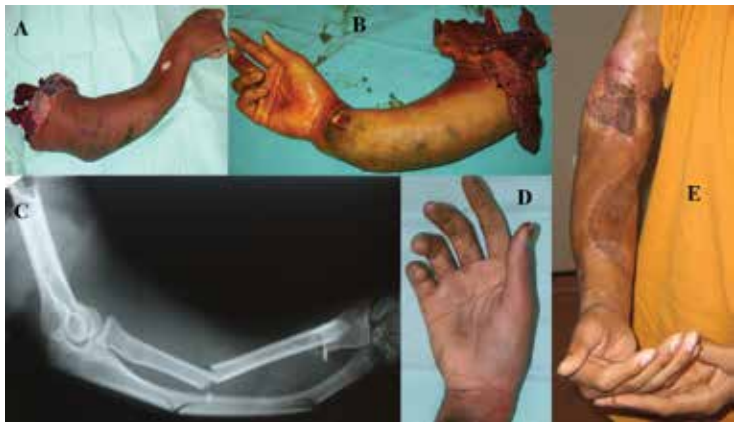

Figure 23. A 17 year old accidentally caught his right arm in a machine used to mix dyes at a carpet factory. **A, B:** He had an avulsion amputation at the mid-humerus level with **C:** segmental fractures of his radius and ulna**. D:** His hand was unharmed though. **E:** Reperfusion was achieved within 4 hours of injury and he attained a good function of his upper limb.

Krapohl et al. [45] showed that a crush injury to the arteries supplying the cremasteric muscle in rats resulted in a significant decrease in skeletal muscle perfusion even though the blood supply though the crushed vessel is maintained and that this may be due to thrombogenic results.

In another interesting animal study, thrombolysis followed thrombosis in rat arteries with induced crush injuries [46]. However, if the crushed arteries were divided and sutured with microvascular anastomosis almost all thrombosed (90%) unless they were irrigated with topical heparin solution which reduced the thrombosis rate but did not promote thrombolysis.

Both these studies show that localised crush injuries to the arteries deserve to be treated with respect and that all such tissue should be excised prior to microvascular anastomoses. It is also important to appreciate that the severity of the crushing may result in amputation of the digits and hand (**Figure 5**). In segmental crushing, however, the distal amputated part, for example, of the hand may be relatively uninjured (**Figure 23**).

3. Conclusion

A thorough understanding of the underlying mechanisms of injury will enable the primary surgeon to pay due diligence where required in detailed planning of the step by step management constantly deliberating carefully between damaged tissues to discard while treating with respect the tissues to be salvaged.

Acknowledgements

Roohi S.A. would like to thank Prof. Dr Lim Beng Hai for his guidance as well as the use of some of his work in the Figures as acknowledged.

Conflict of interest

The authors declare no conflict of interest and have not received any remuneration or benefit from any entity for the writing or publication of this article.

Appendix and Nomenclature

ADM	abductor digiti minimi
DIPJ	distal interphalangeal joint
FDP	flexor digitorum profundus
FDS	flexor digitorum superficialis
K-wire	Kirschner wire
MCPJ	metacarpophalangeal joint
MVA	motor vehicle accident
P	phalanx 1: proximal, 2: middle, 3: distal
PIPJ	proximal interphalangeal joint
PT	post-trauma
ROM	range of motion
RTA	road traffic accident

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Chapter 7

Crush Injuries of the Hand Part II: Clinical Assessment, Management and Outcomes

Roohi Sharifah Ahmad and Pho Robert Wan Heng

Additional information is available at the end of the chapter

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Abstract

This chapter details crush injuries of the hand as opposed to crush syndrome. The definition along with the spectrum of injury is described including a historical review of the causative mechanisms to help in the understanding of basic pathomechanics. The main menu comprises Clinical Assessment, Management and Outcomes, where the reader is taken through the steps one by one on how to approach such an injury, from the history, examination and investigations pointing out important aspects. Basic facts and figures to know and memorize have been placed in an easy-to-absorb format of tables and highlighted boxes. Key points are emphasized, and important aspects of management as well as those simple tips to improve outcome are given to ease the novice as well as the tempered surgeon's encounter. A special section on management of specialized tissue is given toward the end after the basic management is dealt with so that a deeper understanding is gained and applied. Possible outcomes would alert the surgeon on both adverse events to avoid and excellent results to aim for. As always, good functional outcome is sought after but a good cosmetic appearance should be constantly filed away in mind's eye to enhance the final result.

Keywords: crush, hand, injuries, mangled extremity, trauma

1. Introduction

Crush injuries of the hand pose a challenge to even the most accomplished hand surgeons whether it is a minor fingertip injury sustained by getting squashed in a closing door or a high-pressure compression injury involving the palm or wrist.

2. Clinical presentation

The crush injury may present from the tip of the finger to proximally right up to the arm. Although the management of more proximal and therefore potentially life-threatening injuries is precluded from this chapter, we will deal briefly with their management. There are certain principles in the management of all injuries be they minor or major (**Table 1**), and these have to be adhered to, especially if it is a high-energy trauma as in a road traffic accident (RTA) or a fall from a height.

Thus, the primary aim in any trauma is to stabilize the patient and rule out any life-threatening injuries. Airway, breathing and circulation must be first secured. Subsequent management will focus on the limb at hand and its systemic sequelae. After the early treatment and resuscitation of the patient, antibiotic and tetanus cover is ensured and the hand surgeon alerted for further assessment.

2.1. Clinical assessment

The most important part in the management of the crushed hand is the assessment. The crush injury has to be given due respect because neglect in adequate assessment will result in poor functional outcome or unacceptable cosmesis. Thus, a judgment call by an experienced hand surgeon for all tissue components i.e., skin and its contents (tendon, bone, joint, vessels, nerves and muscles), is essential. The aim of the treatment is to provide the hand with basic hand functions most importantly: functioning *pincer mechanism* and *sensation for grasping* (**Figure 1**).

2.1.1. Functional anatomy of the hand

The hand is basically composed of two types of skin, thick palmar glabrous skin and thin dorsal skin. As can be seen in **Figure 1E**, the dorsal skin is thin, loose, mobile and stretchable. The palmar side, however, is thickened for protection with papillary ridges to increase the surface area as well as improve the grip. These ridges form our finger prints. The fingers are highly innervated to provide a two-point discrimination of 4–6 mm at the fingertip, as measured by the two-point discriminator disc (2-PD: **Figure 1F**). The sweat glands give lubrication to aid in feeling and function (**Table 2**).

There are numerous flexure creases to anchor the skin down, and with the palmaris brevis muscle, they aid in holding and gripping objects. The muscles of the hypothenar and thenar eminences allow specialized functions of opponens, abduction, adduction and flexion (**Figure 2**).

- Life before limb
- Limb can endanger life e.g., compartment syndrome, infection
- Look for proximal hidden injuries which can be fatal e.g., head injury, cervical spine injury, associated pneumothorax, intra-abdominal injury
- Thorough assessment—primary survey of the patient as a whole
- Secondary survey of traumatized limb

Table 1. Principles of management of crush injury.

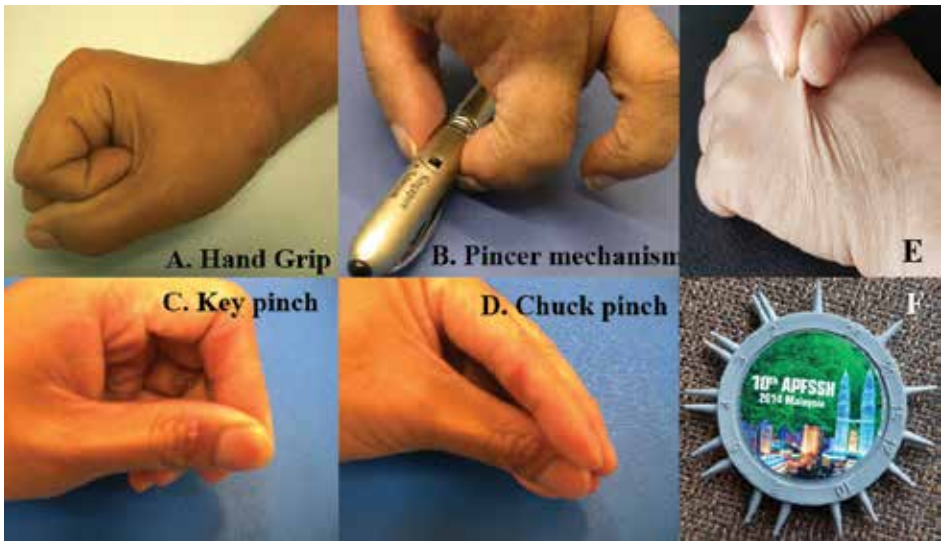

Figure 1. A–D: Of the four basic functions above, the pincer mechanism or "pinch" function is most useful. Pictures taken from Dr. Lim Beng Hai. **E:** Dorsal skin is thinner and pliable. **F:** A two-point discriminator (2-PD) used to measure sensation, which an unfolded paperclip too can do. The patient is first shown the device and explained how it is used to feel one or two points on their fingertip.

2.1.2. History

The injury may be small involving just the digits or the hand, or it may be more extensive including the wrist and even the forearm. It may even be segmental with intervening areas of normal tissue. It may be open or closed, with associated elements of degloving or avulsion. They may be either clean as in door jamb injuries or obviously dirty as the trash compactor or farm injuries. One must also be alerted to marine-related open wounds with their own peculiar pathogens (*Mycobacterium marinum*). It may be contaminated with paint or grease and be sustained in a high-pressure printing press (see previous chapter, **Figure 13**) or injection mechanism (**Figure 3**). Aggravating factors such as friction burns, industrial hot pressure injuries, multidirectional forces and contamination should be assessed carefully.

Palmar skin	**Dorsal skin**
1. Specialized glabrous epithelium	Nonglabrous, hirsute
2. Thick cornified surface	Thin
3. Immobile skin	Mobile
4. High-density sensory receptors	Less dense
5. Subcutaneous pulp	Nail matrix & plate

Table 2. Comparison of specialized tissue in palmar & dorsal surfaces.

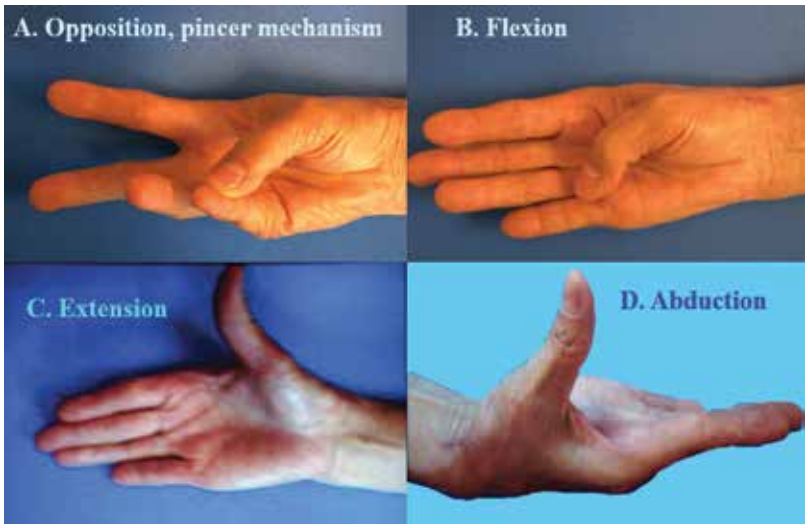

Figure 2. A–D Movements of the thumb: opposition, flexion, extension and abduction. All of these combine to perform circumduction.

The patient's occupation, associated medical conditions and allergies if any, and dominant hand should be recorded. A mental or written note of the patient's expectations would complete the history.

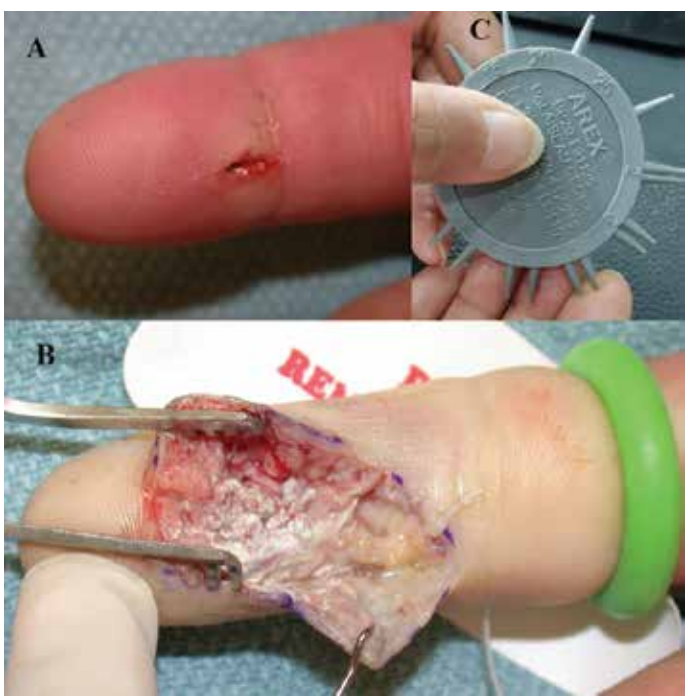

Figure 3. Paint injection injury. **A:** a very small entry point, **B:** but once explored, the extensive damage can be seen. The paint is toxic and needs to be painstakingly cleared. **C:** assessment of nerve function using a 2-PD disc. The test is administered on the normal hand first, then only on the affected digit with the patient's keeping their eyes closed. Just enough pressure needs to be applied so as to lightly blanch the skin.

2.1.3. Examination

Initial assessment in the emergency room can be conducted while obtaining the history from the patient and observing the hand posture, cascade (**Figure 4**), color as well as assessing flexor and extensor mechanisms even without exposing the wound. Local anaesthesia should only be given *after* a sensory examination has been performed, preferably with a 2-PD disc (**Figure 1F**). If delay to theater is expected, a lavage under some local anesthesia maybe performed as necessary. Swabs should be taken if it is potentially contaminated or infection suspected. A more thorough evaluation of the extent of injuries can then be made in the operating theater under general anesthesia and tourniquet control. The assessment should be thorough, with minimum number of wound inspections and made with the surgical treatment options in mind (**Table 3**).

In order to make the choice to replant, reconstruct or amputate, a number of factors have to be taken into consideration (**Table 4**). These deciding factors will influence the surgeon's final decision.

2.1.4. General factors

The patient's general condition is of prime importance in making a decision since an elderly patient in hypovolemic shock would not be able to tolerate further ischemic insult in terms of prolonged operative time and potential blood loss implicated in reconstruction or limb salvage. Similarly, a patient with diabetes would be more prone to infection and complications. In contrast, severe crushing to a contralateral limb would necessitate more extreme measures to attempt salvage. There has been no consensus achieved yet on the maximum time

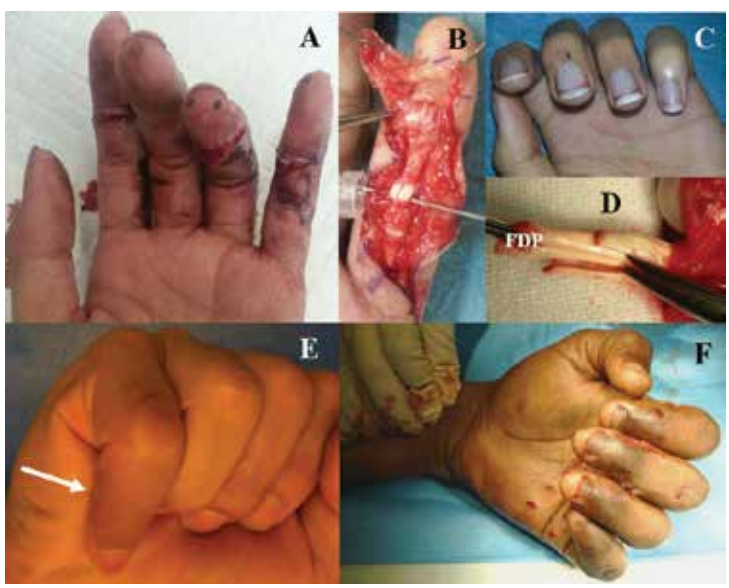

Figure 4. A: Damage to flexor tendons of the index and small fingers can be seen in the flattened posture of those fingers. The index finger cannot be flexed at the DIPJ indicating an intact FDS but a severed FDP (FDS finger). **B:** adequate exposure by gentle retraction with skin hooks and meticulous repair of both FDS and FDP, allows good motion within the sheaths. **C:** restoration of the normal cascade. **D:** the chiasma of the superficialis (tenotomy scissors) opens out and from a volar position proximally, curves 180° to insert dorsal to the FDP on the base of the middle phalanx distally. **E:** in another patient, the small finger cannot be flexed at the DIPJ indicating an intact FDS but a severed FDP. **F:** normal cascade of the hand elicited by the squeeze test.

1. Replantation
2. Resurfacing with skin graft
3. Reconstruct with flap (local or free)
4. Revision amputation
5. Rehabilitation—potential prosthesis/orthosis

Table 3. Surgical treatment options.

General factors	Local factors
1. Age	1. Circulation
2. Circulatory stability	2. Sensation
3. Life-threatening conditions	3. Tendon movements
4. Associated medical problems	4. Loss of skin
5. Injury to contralateral limb	5. Loss of bone & joints
6. Time lapse—warm & cold ischemia tissue	6. Loss of specialized tissue
	7. Mechanism of Injury

Table 4. Assessment of general factors & local factors.

an amputated part of a limb can tolerate ischemia and gain good functional postreplantation. Many factors are involved such as ambient temperature, collateral circulation and level of amputation. Many attempts have been made to assist in this decision-making process but none are ideal. Although MESS (**Table 5**: Mangled Extremity Scoring System) was initially developed for the lower limb [1], it has subsequently been applied to the upper limb as well with some degree of success, provided one knows its limitations [2, 3]. In this score, a total of six implies the limb is salvageable, while seven or more advocates amputation. The score, however, does not take into consideration other major injuries causing hypovolemia, associated medical conditions, injuries to the other limbs that may necessitate salvage, nerve injuries and the type of damage sustained, and a more subdivided range of ages. The latter is important in that an infant would have better nerve regeneration capabilities than an adult, whereas a 90-year-old would have less cardiovascular reserve than a 50-year-old, both situations not being differentiated. The exact mechanism of injury (crush, avulsion, guillotine), as well as the amount of force, its velocity & the width of the offending object are all important factors in decision-making. Smith et al. suggested three contraindications to major replantation, namely brachial plexus avulsion, severely mangled extremity and an excessive ischemia time [4]. There have been many advances since then to overcome some of these obstacles, for example, highly innovative developments in the field of neuromuscular prosthetics; however, brachial plexus avulsion remains challenging. There will come a time though when an equilibrium will be reached in the two arms.

Skeletal/soft tissue injury	
• Low energy (stab, fracture, civilian gunshot wound)	1
• Medium energy (open or multiple fracture)	2
• High energy (military gunshot wound, crush)	3
• Very high energy (+gross contamination)	4
Limb ischemia	
• Pulse reduced or absent but perfusion normal	1
• Pulseless, diminished capillary refill	2
• Pt is cool, paralyzed, insensate, numb	3
Age	
• <30	0
• 30–50	1
• >50	2
Shock	
• Systolic BP always >90 mmHg	0
• Systolic BP transiently <90 mmHg	1
• Systolic BP persistently <90 mmHg	2

Table 5. Mangled extremity severity score.

2.1.5. The five P's

The most important aspect in the assessment of the limb will be the *vascularity*. Traditionally described as the 5 P's namely:

- Pain
- Paresthesia
- Paralysis
- Pallor
- Pulselessness and additionally a
- Cold limb

Pain, paresthesia and paralysis may not be detected in the head injured patient, patients that are heavily sedated, or those with brachial plexus injuries. Pallor may be unreliable in certain situations such as hypovolemic shock, massive blood loss, pigmented individuals and compartment syndrome. Dyes and other coloring materials as well as grease have to be scrubbed off in order to properly assess the circulation. Other parameters such as capillary refill, bleeding on pin prick, and transcutaneous oxygen pulse pressure may be used as an adjunct in the assessment.

2.1.6. Specific structures

Neurological damage also has to be outlined prior to exploration and also to prepare the surgical field in case a nerve graft is required. Gross sensation being inadequate, two-point discrimination should be used whenever possible to ascertain neurological deficit. Failure to differentiate 8 mm is the cutoff point where damage is certain.

Assessment of **tendons** can be tricky, especially if an adjacent nerve is also damaged or if the injury is closed. The best way to assess tendon damage is to observe the **resting posture** of the hand (**Figure 4C**). The unmistakable cascade of increasing flexion from the index to the little fingers is lost if there is any incontinuity. Complete division of both flexor tendons to the digits results in a straight finger in comparison with the adjacent flexed digits (**Figure 4A**). Division of the flexor digitorum profundus (FDP) straightens out the DIP joint (**Figure 4E**). A cut flexor digitorum superficialis (FDS) alone may not produce any visible abnormality (**Figure 4A**); however, a partial tear may be diagnosed by eliciting pain while stress testing the affected tendon. In a child or semiconscious adult, flexion may be reproduced by squeezing the forearm volar surface (**Figure 4F**). All these subtle differences should be looked for in the initial examination. **Flexor tendons** are tough structures and usually the last to be severed. Nonetheless, they are not easily replaced, having an intricate anatomy and biomechanism not completely reproducible. Therefore, it follows that they should be debrided with care.

Extensor tendons may be partially or totally cut presenting in a spectrum from a closed mallet, to just weakness in extension, or complete inability to extend the finger. Extension should be tested against resistance and if weak may indicate either a partial tear, damage to the collateral bands, or a single tendon cut in a dual tendon finger such as the index or little fingers. Avulsion of the central slip may present as a boutonniere deformity (**Figure 5**). Patency of the central slip is determined by Elson's test, where the finger is tested against resistance with the PIP is flexed 90° over the edge of a table and resistance applied to active extension [5]. If the central slip is avulsed, the PIP extension will be weak and the DIP will be rigid [6].

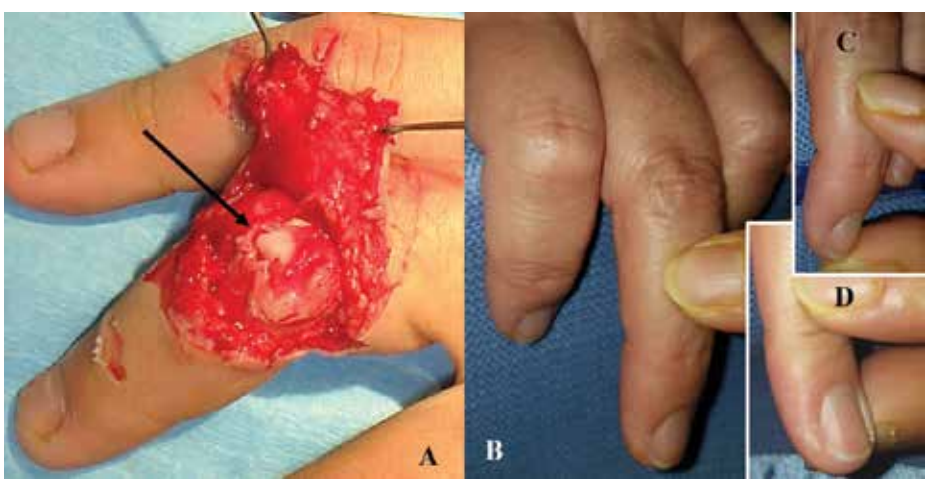

Figure 5. A: The central slip is torn (black arrow), and this incontinuity will result in difficulty for the PIP joint to be extended. **B:** The finger can still be extended (weakly) through the lateral bands, which are intact and inserted via the terminal tendon into the distal phalanx. **C:** If the central slip is intact; however, the P3 will be "floppy" **(D)** While extending the PIPJ in flexion. See video by Dr. Mike Hayton: https://youtu.be/G9HY0qXWUvE [6].

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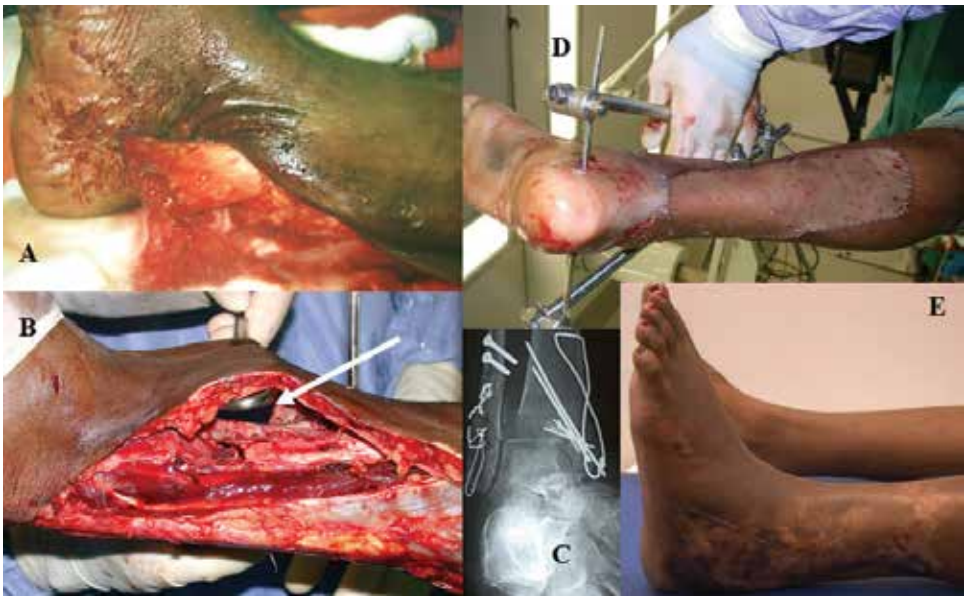

Figure 6. A:A 35 year old sustained a highly comminuted fracture of the distal end of her tibia and fibula. **B:** She also had a *severe degloving injury* (arrow) with partial loss of skin cover. **C:** Meticulous reconstruction with wires, screws and basic principles. **D:** Stabilization with an external fixator and skin graft posteriorly. **E:** Good healing 3 months later with relatively normal gait.

Skin is assessed both from the history and then examination. A roller or roll-over injury suggests degloving (**Figure 6**), which can be more extensive than the wound implies. Bleeding from the wound edge and to pin-prick, color of the skin and separation from the underlying subcutaneous plane all play an important role. If the color is pink with active bleeding and a rapid refill, circulation is intact. If the color is slightly dusky, and the refill is rapid but bleeding is dark in color, there may be venous congestion. If, however, the skin is pale and refill is poor, revascularization may be necessary. A note is made to the patient and his family, of the possible flaps, both local and distant that may be required.

If **radiographs** from the emergency room are not satisfactory in assessing the **bony** injury, it may be necessary to repeat them either after splintage or in the operating room This would aid in assessing the damage accurately and also in planning the implants to be used in the repair or reconstruction of the skeletal framework. Ideally, anteroposterior and lateral films should be available, and preferably oblique as well if the fracture configuration is complex. Torsional injuries may require reduction and splintage in position prior to radiographic evaluation. It may save time to order chest and cervical radiographs for the preoperative assessment if the patient is older and general anesthesia is required for surgery.

3. Management

3.1. Preoperative assessment

After a relevant detailed history and thorough examination, the patient is worked up for the operative procedure. The assessment is targeted to evaluating his general condition and co-morbidities

if any, and locally to the injury concerned. The pre-operative laboratory tests would depend on the hospital policy, the patient's age and associated medical conditions if any. Thus, while the older patient (above 40 years of age) would require a chest film, electrocardiogram, full blood count, glucose levels and renal function, the younger patient may do with just the blood counts.

3.2. Surgical treatment

The primary aim of management is to save life, limb and then restore limb function in that order. After that, the aim must be to achieve a function better than that offered by a prosthesis. The principles of replantation and hand reconstruction (**Table 6**), however, are very stringent and demanding for there is little room for error.

3.3. Radical debridement

The hand surgeon's greatest enemy is infection secondary to inadequate debridement and lavage, cited by Wang et al. as the most common cause for failure of survival [7]. It follows therefore that the most important step after the initial assessment is meticulous and thorough debridement of all devitalized tissue and foreign bodies to convert a dirty contaminated wound into a clean surgical wound.

Prior to starting surgical debridement, the wound should be adequately irrigated with copious amounts of saline, povidone-iodine (Betadine) and hydrogen peroxide. Some authors have advocated a concoction of the three in a ratio of five parts to one to one, respectively [8]. Washing with gentamycin either neat or diluted with saline is also practiced by some, although clinical support for this is not convincing. Pulsatile lavage has also been advocated as has rigorous scrubbing with a brush. Ultimately, the aim is to get the wound clean *with minimal amount of tissue damage*.

Tomaino said "débridement must be consummate, as if ablating tumor" [9]. Therefore, shredded tendon pieces and avulsed nerve without axonal elements must be excised. Similarly, bone pieces without any muscular or tendinous attachments should be removed. Although difficult to replace some tissue types (such as tendon, nerve and joints), retention of these contaminated or devitalized tissues may result in more harm than good. Whether to debride under tourniquet control or not is dependent on surgeon's preference. No doubt, the surgical field is much clearer with the tourniquet up, but one has to consider tourniquet time, especially if it is a replantation.

- Radical debridement to convert dirty to clean surgical wound
- Obtain a stable skeletal scaffold
- Good soft tissue repair and reconstruction
- Adequate skin resurfacing, soft tissue cover
- Rehabilitation and mobilization to restore function and early return to work
- Cosmetic acceptability by the patient
- Psychological support

Table 6. Principles in hand reconstruction.

Preferably, the skin edges should be débrided with a sharp instrument such as a blade without the tourniquet to assess skin, subcutaneous, muscle as well as bony viability. When it comes to fine dissection and meticulous débridement; for example, around vessels and digital nerves, the tourniquet could be inflated for that particular period. Of course, preferentially, tourniquet control is used for repair of bones, arteries & veins, tendons, and nerves in that order. The use of diathermy should be restricted and drains avoided (**Table 7**). The wounds should not be closed under tension, and if highly contaminated, delayed primary closure is entertained. If in doubt, a second look and repeated débridements should be carried out. Emergency free flaps have been found to have a higher rate of success with fewer numbers of procedures. They prevent desiccation of tissue & reduce infection, thus ultimately improving the function and eventual outcome [10].

3.4. Management of specialized tissue

Once the wound has been thoroughly débrided, a secondary inspection should be carried out to assess the damage sustained and the best course of action to be undertaken. Reid classified mutilating injuries of the hand into (1) dorsal injuries, (2) palmar injuries, (3) radial hemiamputation, (4) ulnar hemiamputation, (5) distal amputation, and (6) degloving injuries [11]. Das De et al. preferred assessment of injuries to be based on damage sustained in terms of tissue components—bone & joints, tendon, muscles, nerve, vessels, and skin—involving different anatomical parts [12]. Although various authors have used different anatomical classification areas (e.g., Tomaino—dorsum of hand, volar forearm and thumb-index web [9]), we feel division into digits, midhand, wrist and forearm is simple and useful both anatomically as well as functionally. The various tissues will still have to be considered individually in each of these areas.

3.4.1. Skeletal stabilization

Bones provide the stability and skeletal framework to which the soft tissues are attached while joints offer mobility to the hand and digits. When damaged severely as in crush injuries, the hand becomes flail and thus useless and repair of the skeleton has to be undertaken before soft tissue reconstruction proceeds. It is therefore essential that fracture fixation is adequately performed primarily and is *strong enough to provide a stable scaffold to undertake further soft tissue repairs as well as undergo rehabilitation*. The issue of internal versus external fixation is not so much of a dilemma in the upper limb as it is in the lower limb and decision-making is

- Adequate lavage, with saline, gentamycin or H_2O_2
- Reduction in the prolonged use of the tourniquet
- Sharp debridement and meticulous tissue handling
- Minimize diathermy
- Avoid wound closure under tension
- Leave wounds open if heavily contaminated
- A second look and repeated debridements after 48–72 h
- Emergency free flap if required

Table 7. Guidelines to achieve satisfactory debridement and closure of wounds.

relatively straightforward (See previous chapter, Figure 22). The upper extremity is blessed by a better blood supply and less infection. Grade IIIB and IIIC fractures are common but deserve respect and caution. Whether to use plates, screws or Kirschner wires depends on the amount of contamination, bone loss, soft-tissue defect, tendon system and rigidity desired. Of course, surgeon's preference and the time factor play an important role too. There are now a compendium of internal fixation devices of which we find the low-profile variable angle locking plates very handy (**Figure 7**) [13]. When there is a soft tissue deficit, implants may be exposed, but if cover can be achieved within 5–7 days, Godina believes infection is usually not a problem [14]. Bone loss, however, preferably should be grafted within 8–10 days, so long as a clean surgical wound has been created after adequate debridement [15].

Sometimes in severe comminution in the **digits**, closed pinning with cross K-wires has been found to be quite useful, since the intact periosteal sleeve provides support. Some surgeons believe, however, that K-wires cause "spot-welding" of tendons, especially on the dorsum of the hand, pinning down the extensor mechanism. Care should be taken in placement of these wires, which should preferably be buried under the skin and away from tendinous or ligamentous structures for ease of hand rehabilitation as well as reduction in infection rates (especially in tropical climes). They can be removed later if need be rather than earlier if they are subcutaneous.

Severe damage to the **DIPJs** is fairly well tolerated by fusion, but with the advent of the miniscrew (1.0 1 nd 1.2 mm), tiny fractures may be dealt with precisely. The hook plate by Medartis® tackles the difficult dorsal lip avulsion fracture beautifully; however, the sequence of performing the fixation is key and care should be taken not to injure the germinal matrix (**Figure 8**). **PIPJs**, however, are a different matter. Every effort should be made to salvage the PIPJ if feasible especially in a badly mutilated digit. The articular surface should be painstakingly reconstructed with interfragmentary microscrews, cerclage wires, K-wires, or even the hook plates (**Figure 9**). Stable fixation will allow early mobilization and achieve better results.

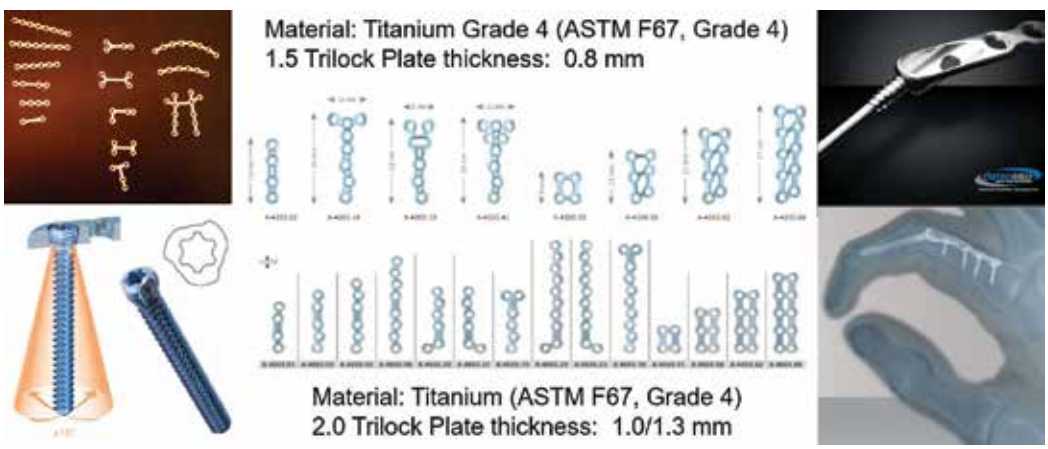

Figure 7. A wide array of titanium low-profile plates of various sizes and shapes are now available to suit the hand surgeon's and patient's needs. Top left: the older miniplates. Left: the trilock mechanism—a neat way to get the screw head to lock in the plate. Middle: the Medartis® range—one of the most "low-profile" plates in the market with chamfered edges for both plates and screws. Right: the Osteomed® PIP and MCP fusion plates—a novel development.

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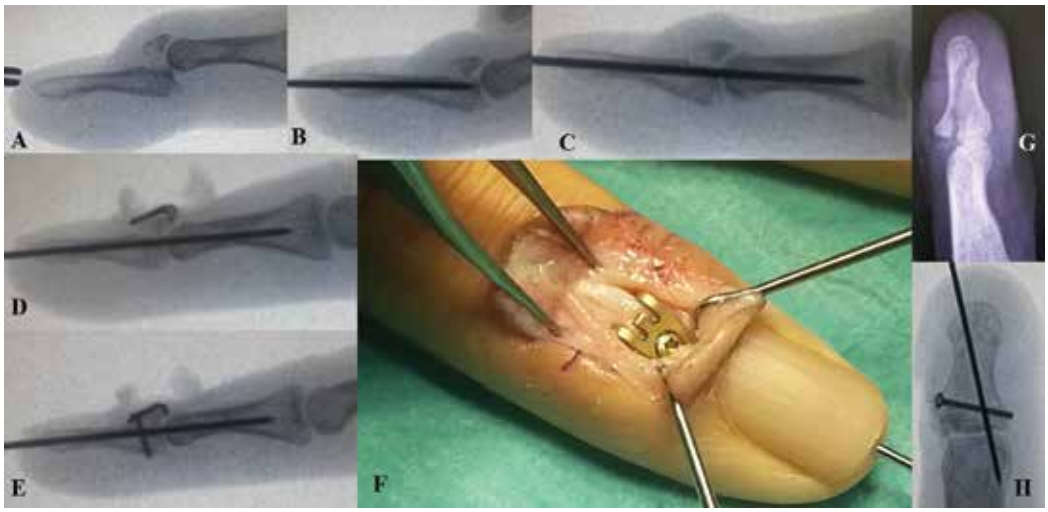

Figure 8. A: there is an avulsion fracture of the dorsal lip of the distal phalanx (P3) and the DIP joint is subluxed volarly. **B:** a central 0.8-mm K-wire is inserted in to P3, the joint is reduced and **C:** the wire is pushed into the middle phalanx (P2). **D:** the approach is then made dorsally and the fracture fragment reduced to P3. An incision made in the germinal matrix prevents it from being compressed. **E:** the holding screw is inserted. **F:** final picture. **G:** a severely crushed P3 with mallet deformity requiring bony stability was fixed with a 1.0-mm miniscrew. **H:** postoperative film.

Having said that, however, we have had success with an intra-articular pylon fracture treated nonoperatively with ligamentotaxis (**Figure 10**). **MCPJs** tolerate fusion to a better degree than PIPJs, but it is rarely required for they adapt quite well to marked degrees of malpositioning. Basal **metacarpal** fractures may be stabilized by transverse K-wiring to maintain height unless it involves the ulnar base of the fifth metacarpal (attachment of the ADM), where

Figure 9. A: intra-articular fracture of the radial condyle of P1 of the left MF. **B, C:** reduction and fixation with a 0.6-mm K-wire and a 0.4-mm cerclage wire. **D, E:** good grip and ROM—10–110°. **F, G:** a 23-year-old engineering student presented a month later with a volar lip fracture (P2) and dorsal dislocation of his R ring finger. **H, I:** through a volar approach, the two condylar fragments were reduced and held with K-wires before miniscrews were inserted. **J:** the volar plate was reattached with a bone anchor suture. At 3 months postoperation, he had a 20°-extension lag and full flexion.

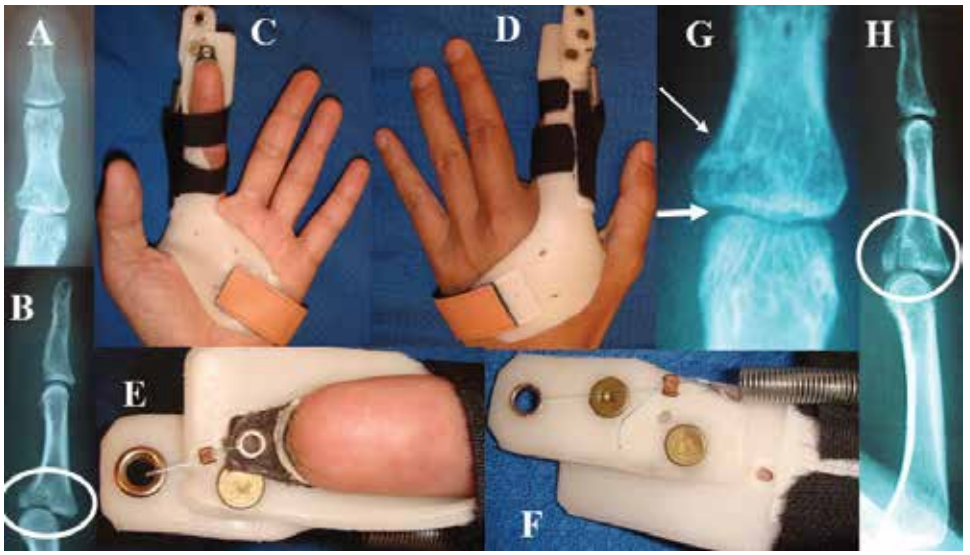

Figure 10. A, B: a die-punch type of pylon injury to the base of the middle phalanx (P2). The patient was not keen on surgery. **C, D:** overall view of the thermoplastic splint. **E, F:** close-up view. A strap was glued to the dorsal nail plate and traction applied. **G, H:** the fracture reduction as seen after 3 to 4 weeks.

fixation ensures no displacement (**Figure 11**). Fractures of the neck of the fifth metacarpal deserve special mention because of the associated stiffness that develops in the MCP joint. Cross K-wiring is good in experienced hands, however, unpredictable in terms of adhesions, so we advocate low-profile plating and early mobilization (**Figure 11**). Metacarpal **diaphyseal** fractures, on the other hand, may require either intramedullary fixation or plates and screws. In spiral fractures, two interfragmentary screws or even cannulated microscrews (**Figure 12**) are warranted for there is a likelihood of shortening. In stabilization of transverse or comminuted fractures, especially in multiple segments and multiple digits, however, *rotation of digits should be given special attention*. Axial and especially rotational malalignment is magnified from proximal to distal starting at the metacarpal level, thus plating is a better option and little added time is necessary. Prevention is the key, by checking rotation after the fixation. The present minihand plates are lower in profile, chamfered (including the screws) and even locking, causing little interference to the tendons and providing the required rigidity. If there is a significant amount of bone loss, the normalization of length can be achieved by transfixing K-wires through the adjacent metacarpals and bone grafting the defect plus plating if the gap is up to 8 cm long (**Figure 13**). Another option, which is useful in replantation surgery, toe transfer or transverse fractures, is intraosseous 90–90° wiring, be it digital or metacarpal level (**Figure 14**). This may also be used to fuse the DIPJ or the PIPJ, as modified by Lister with one intraosseous wire and a transfixing K-wire [16]. The MCPJ can also be fused using a plate and screws: a special set is available from Osteomed® [17] (**Figure 7**). Fusion of a joint primarily has to be thought of carefully and prior discussion with the patient is essential. The angle of fusion would depend on the patient's vocation, age, the dominant hand, involvement of adjacent joints, in the same digit, the neighboring digit and the other hand.

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Figure 11. A: intra-articular fractures of the bases of the fourth and fifth metacarpals. **B:** a low-profile ladder plate from Medartis® is used to attain precise reduction. **C:** a cannulated screw and K-wires were only possible for the fifth base due to extensive comminution. **D:** another patient with basal fracture dislocation of the fifth metacarpal and CMC joint. **E:** the basal K-wire was used to reduce the fracture fragment, but it started to push it away, so the proximal K-wire was used to maintain height as well as push the fifth metacarpal toward the fourth and hold it in place. **E, F:** a square plate was applied on the dorsal surface straddling the joint with screws in the hamate to keep reduction and immobility. **G, H:** patient with a very distal fracture neck of the fifth metacarpal. **I:** the miniplate applied (1.5 mm Medartis® Aptus) allows a full hand grip without interfering with the tendon mechanism reducing fifth MCPJ stiffness.

Forearm fractures present a challenge since they need to be internally fixed in order to achieve bony stability. In forearm fractures where open fractures are more common than closed [18], Gustillo type I, II and IIIA are safely fixed internally, while with IIIB and IIIC fractures, there is still debate [19]. Early internal fixation (within the golden 6-h period) seems to be acceptable, but patient selection is still important and if in doubt, external fixation is advised, converting 8–10 days later to internal devices when bone grafting can also be done [20].

3.4.2. Tendon repair and reconstruction

Flexor tendons are special because they are not only intricately entwined (**Figure 4D**) but they are housed in a unique tunnel providing nutrition, a smooth gliding environment and a bio-mechanical advantage. This pulley system is as important as the tendons themselves and any damage to the A2 and A4 pulleys should be repaired or reconstructed. Tendon repair should be tension free and fairly good quality tendon should be used. The advantage of a strong repair and early mobilization over a tenuous repair and cautious rehabilitation cannot be over-emphasized.

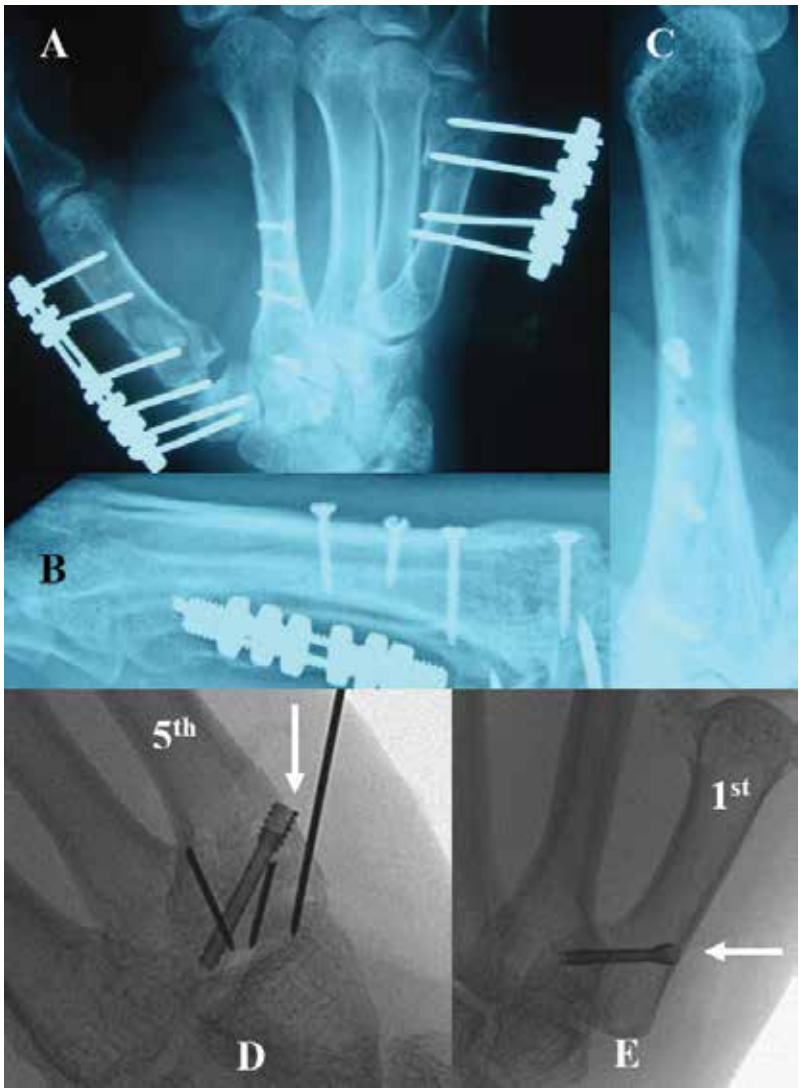

Figure 12. A: very comminuted fractures of the first and fifth metacarpals, while the second (**B, C**) was relatively amenable to minimal screw fixation because it was a long spiral. Four screws were required! **D:** a basal fracture of the fifth metacarpal with considerable comminution addressed by a cannulated screw (white arrow) and K-wires. **E:** similarly a single cannulated 2.2-mm screw (white arrow) correctly placed can tackle the volar beak fragment of the first metacarpal fracture.

A strong repair constitutes a 4/0-sized core suture (four or better still six strands) with an epitendinous 6/0 suture, which usually provides 20% of the strength. The key is to balance the number of core sutures (strength) versus overstuffing the flexor tendon and adversely affecting its blood supply [21]. Another point to note is if the technique is too complicated, repeated grasping of the tendon (should only be held once with an Adson toothed forceps) would cause damage. If the tendon is frayed, attenuation and gapping may occur inevitably resulting in delayed rupture and less than optimal results. A tendon gap may be bridged by

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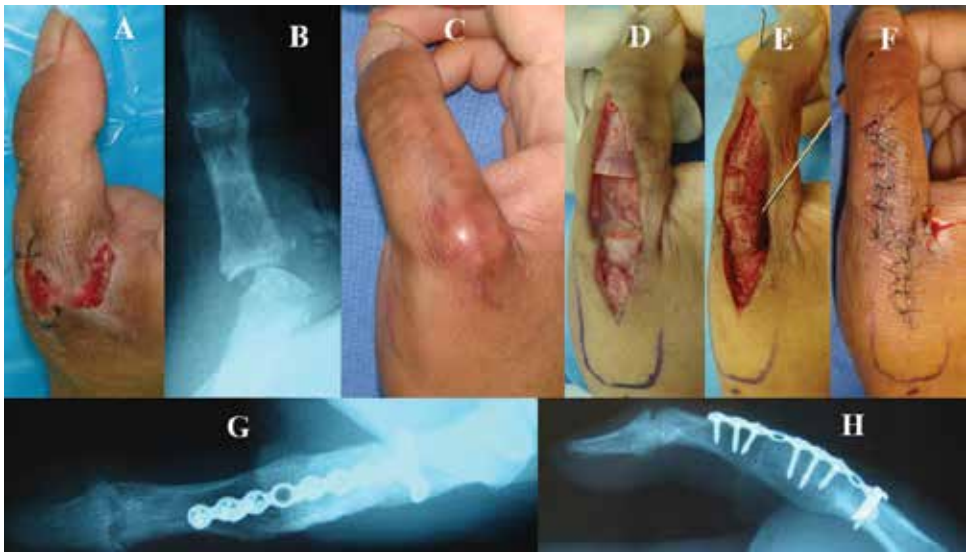

Figure 13. A, B: an infected wound after a snake bite destroyed the proximal phalanx (P1) of this gentleman's right thumb. **C:** once the infection cleared, the affected thumb was pronated, dislocated and nonfunctional. **D, E:** a 2-cm segment was removed and replaced (fused) with a bone graft and plated, **F:** in the correct position.

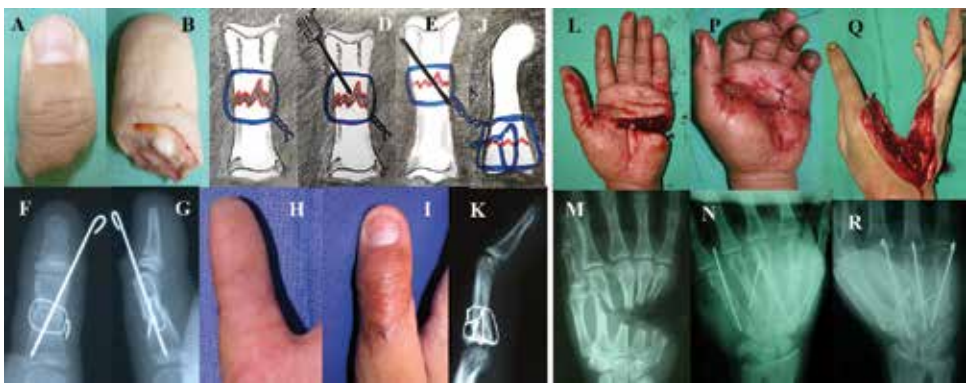

Figure 14. A, B: a complete amputation of the right thumb in a 10 year old. **C:** the cerclage wire (0.4 mm) is placed in position around the fractured bone ends as shown and twisted. **D:** an appropriately sized cross K-wire is then passed through the first cortex but not through the fracture site. **E:** the bone ends are approximated and the wire tightened (a drill may be used for this) with care so as not to break it. The K-wire is then pushed through as reinforcement. **F, G:** on x-ray. **H, I:** clinical picture at 3 months. **J, K:** if no K-wire is used but two cerclage wires are placed at 90° to each other. **L, M:** a transverse transmetacarpal partial amputation. The metacarpal area has a lot of structure packed tightly together making this a challenging surgery. Minimal and rapid fixation is key to avoid muscle ischemia as well. **N, P:** Lister's wiring may be used or **Q, R:** cross K-wires.

an intercalary tendon graft, but extensive damage with resultant tendon loss especially after debridement is best treated with tendon transfers or two-stage reconstruction. If a flexor tendon is forcibly approximated, or the pulleys are by-passed, contracture is almost inevitable (**Figure 15**).

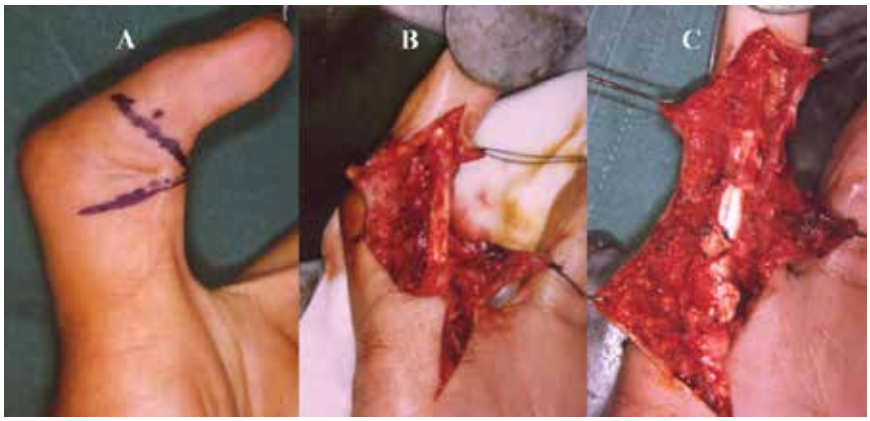

Figure 15. A: 17-year-old boy was referred for a flexion contracture of the PIPJ by a district hospital. **B:** the surgeon had done a repair of the FDP tendon outside the pulleys, resulting in "bowstringing." **C:** a reconstruction of the pulley system was done using one slip of the FDS tendon.

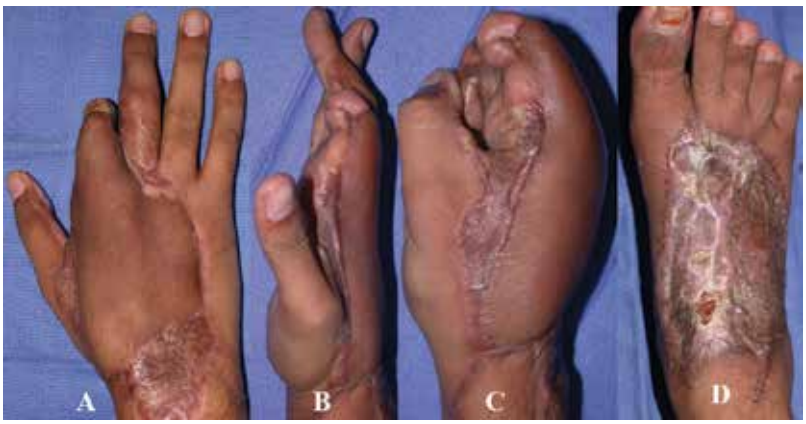

Figure 16. A 25 year old used the dorsum of his right hand to stem his slide on the road from a motor vehicle accident—"brake pad" injury. **A:** dorsal defect exposing second metacarpal, proximal, middle and distal phalanges was covered with a dorsalis pedis flap. **B:** active extension achieved in the index with the extensor digitorum brevis graft anchored to the distal part of the middle phalanx. **C:** a reasonable grip. **D:** the donor defect is not cosmetically pleasing even in a young healthy patient.

Extensor tendons are flatter and more superficial also requiring a favorable soft tissue environment for gliding. Thus, extensive dorsal soft tissue defects with tendon loss should also be treated with tendon reconstruction be it either with a composite graft as in a dorsalis pedis flap (**Figure 16**) or a tendon graft in a free flap. Larger tendon gaps are also not well tolerated if primarily repaired. They cause shortening and alter the biomechanics of the extensor mechanism. The dorsal capsule (wrist) may be used to bridge smaller defects of less than 3 cm (one phalanx). A minimal gap that can be repaired without undue tension, usually gives good results, and is more forgiving than the flexor side. Sometimes even closed management with splintage gives good results as in a closed mallet.

Distal anchorage of tendons can be difficult and various ways have been devised to overcome the problem. The steel suture with a button is traditionally used for zone 1 FDP injuries.

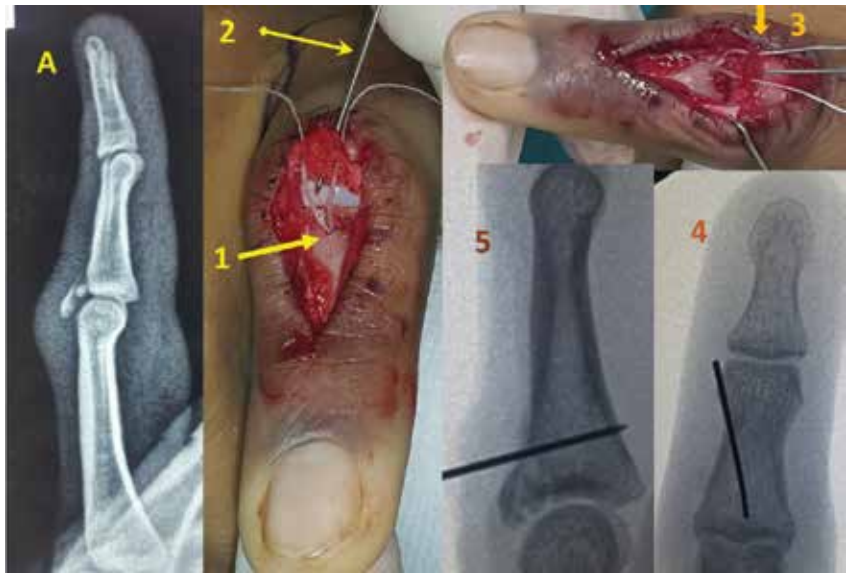

Figure 17. A 25-year-old male skidded off his bike and sustained an extensor central slip avulsion fracture of his right index finger. (1) The first step is to lift off the avulsed fragment and centrally place a bone anchor at the base of the middle phalanx (P2). We used a JuggerKnot soft Anchor 1.0 mm® [23]. (2) An appropriate K-wire is then used to hold the avulsed bone fragment. (3) The suture needles are carefully threaded through either side of the fragment taking care not to fracture it further! (4) The fragment is reduced distal to proximal against the pull of the tendon. (5) K-wire is anchored through volar cortex.

Newer methods include mini-Mitek screws with swaged sutures for both flexor and extensor tendons [22]. These provide firm anchorage to the proximal phalanx, carpal bones and small structures via a 1.8 or 2.0 mm screw and sutures swaged to it to reattach the severed tendon. A very recent product in the market is the absorbable JuggerKnot® soft Anchor 1.0 mm mini where there is no metal implant hence no chance of irritation to adjacent tendons (**Figure 17**).

3.4.3. Muscles

Crushed, contused, contaminated and frayed muscle fibers should be excised to prevent infection. Retention of these would cause fibrosis, contracture and stiffness [24]. Ischemic muscle similarly should be excised. This problem is frequently encountered with the intrinsic muscles of the hand, and special thought should be given intraoperatively since inadequate excision will subsequently interfere with tendon excursion. A "burst laceration" of the first web space also demands *wide excision* and perhaps prophylactic release of certain compartments (see previous chapter, **Figure 19**). A close watch for nerve compressions and compartment syndrome should be instituted with anticipated repeated debridements.

3.4.4. Nerve

Of all the tissues that are damaged, nerve recovery is the worst, even in the best of circumstances. We have made advances in the repair and reconstruction of bone, soft tissue and vessels, but nerve repair remains a challenge. Perhaps biological options may be the way to go in the future—these are promising results [25]. At present, a clean cut nerve in a young patient with the best repair

possible may still not recover full function. The functional outcome of nerve repair depends on the **age** of the patient, the nature (**mechanism**) of the injury, the **distance** from the end organ and any **associated medical** conditions. The younger the patient, the better the outcome, and more aggressive methods may be attempted, such as nerve grafting or using a venous conduit to bridge a gap [26]. In older patients, and those with associated ailments, it is best not to await nerve regeneration, especially if it is a proximal injury. Tendon transfers may be considered at an earlier date, and even primarily. A clean cut injury to the nerve has a better prognosis than crush injuries, which are better than avulsions. If the wound is contaminated or there is a wide gap, it is better to perform delayed nerve grafting. The **nerve involved** also plays a role in functional outcome, the radial being more forgiving as compared to the ulnar, with the median in between. In the fingers, (with the exception of the little finger), the radial digital nerve is preferentially repaired over the ulnar where both cannot be repaired and one has to be sacrificed. In the thumb although both are important, the ulnar digital is essential for pinch grip. In the event of a missing segment, nerve grafts may be harvested from cutaneous nerves in the antecubital fossa.

3.4.5. Vessels

Once skeletal stability is obtained, repair of arteries should take precedence over the veins especially when ischemic time is of importance. This may not be evident in the digits, but in the **hand, wrist and forearm**, it becomes crucial. In **major replantations** and revascularizations, even bony stability may have to be sacrificed in the interest of ischemic time and early reestablishment of circulation. This comes at a cost of subsequent damage to the repair. Alternatively, shunting has been used to decrease the risk of metabolic toxicity upon revascularization and also accumulation of these toxins in the limb by mechanical washout and chemical nutrition. Some have tried free radical scavengers in this process to reduce the ischemic-reperfusion injury (as cited by Shatford and King [3]). In segmental injuries, it may be best to use an interpositional vein graft to achieve reperfusion across the zone of injury.

In the **hand**, blood supply can be adequately assessed by clinical examination. There is little use for angiography and time is of the essence. If a pedicled regional flap based on the ulnar or radial artery is being planned, however, then it may be warranted. Usually, the hand is well perfused with the distal and proximal arches and numerous collaterals. Damage may cause some ischemia that may be noticed only later on in the intrinsic musculature.

In **digits**, the dominant digital artery is usually the radial digital in the little & ring fingers and the ulnar in the index and thumb. These again are preferentially repaired. Repairing one artery and one vein improves circulation to all the ischemic tissues and aids healing. Thrombosed and contused vessels (arteries) should be adequately resected and bridged by an interpositional vein graft if revascularization is indicated. Failure of the digit to pink up after repair should alert the surgeon to check on the patient's hypovolemic, hypothermic and peripheral circulation before proceeding to check the anastomosis, proximal pathology and distal vessel or end organ damage.

3.4.6. Skin and subcutaneous tissues

"Tendons, nerves, joints and bones cannot be left exposed to the outside world and when infected cause sloughing, purulent arthritis, osteomyelitis, months of dressing and final

crippling. All vulnerable parts should be protected from such fate by covering them over and the wound closed, thus saving months of invalidism, compensation and industrial waste, and big permanent disability." These prophetic words written by Sterling Bunnell in Surgery of the Hand in 1944 still stand true to this day.

3.4.7. Resurfacing options

In resurfacing a loss of skin and subcutaneous tissue in the hand, the choice option will depend on the **extent** of skin loss, the **site** involved including the **specialized tissue** lost and the personal preferences and **skills** of the surgeon.

The reconstructive ladder (**Table 8**) as referred to by Levin was designed from the simplest option to the technically most demanding [27]. The best option, however, still has to consider the above-mentioned factors and may differ from surgeon to surgeon. With a small sized wound of less than 1 cm^2, **epithelialization** takes about as much time (2 weeks) as **primary closure**, hence the former is usually chosen. **Skin grafting** has specific requirements, like a healthy bed with adequate blood supply. It cannot be placed on exposed tendons or bone and does not provide sensation. It is not as durable and contouring becomes difficult over the web spaces. Sometimes the damaged skin when healed causes a contracture, which may be dealt with by z-plasty, especially to deepen the first web space or to release a volar flexion contracture in the digits (**Figure 18**). In the first web space, we have found the Ostrowski flap to be particularly useful (**Figure 19**).

A **local flap** garners its blood supply from the surrounding tissue and hence may not be suitable if the bed is unhealthy and sources outside the zone of pathology are preferable.

The **pedicled flap**—so-called because it is based on a named artery—although away from the injury area and does not require microsurgery, has its limitations in size and reach. Besides, donor sites are limited and donor morbidity can be high. However, we have found homodigital neurovascular island flaps, cross finger flaps, reverse digital flaps and posterior interosseous flaps to be useful and safe flaps to cover hand and digital skin losses. The kite flap described by Foucher [29] leaves a prominent scar on the dorsum of the hand (**Figure 20**) but a slight modification as in the radial transposition flap described by Rae and Pho results in a cosmetically pleasing fine line, which can be placed on the radial border of the index [30].

Epithelialization
Direct closure
Grafting—split or full thickness
Local flap
Pedicled flap
Free flap

Table 8. The reconstructive ladder.

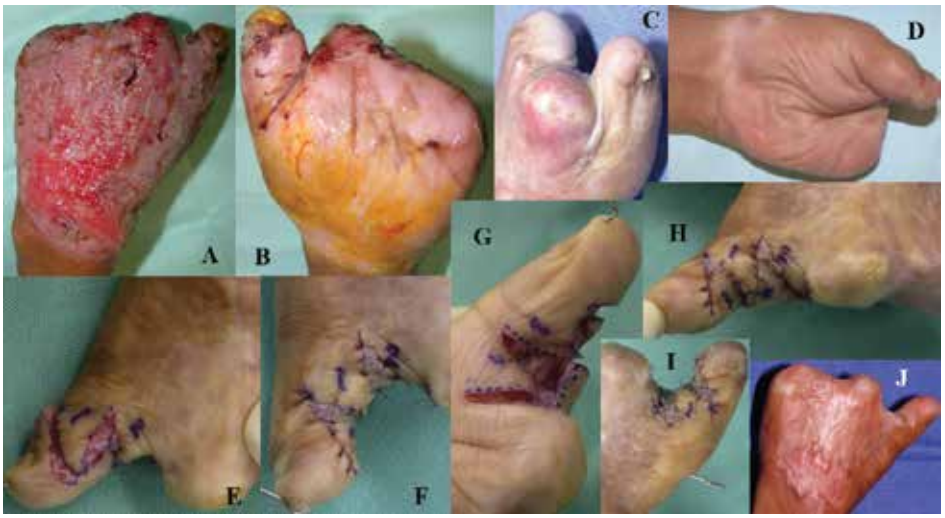

Figure 18. A, B: two days post-trauma (PT), the patient ends up with a metacarpal hand and a partial take of the thumb replantation. **C, D:** 6 and 9 months PT—there is no pincer grasp. **E, F:** the dorsal and volar designs of the Z-plasty flaps. **G, H:** the flaps in place. **I:** held in abduction with a K-wire. **J:** final result at 18 months PT.

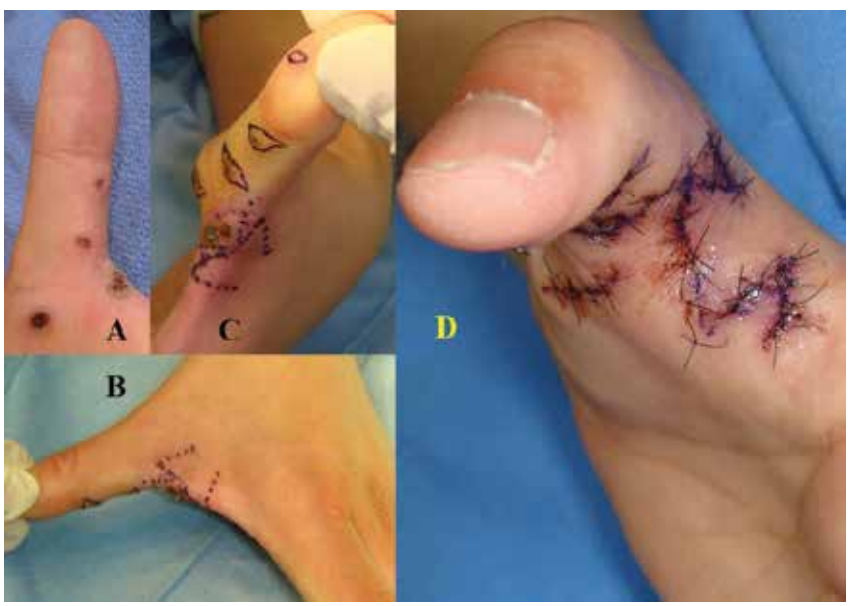

Figure 19. A: a child with multiple viral warts not responding to conservative treatment. **B, C:** due to the placement of the lesions, it was necessary to perform a flap to prevent later contracture of the first web. **D:** the Ostrowski flap was thus designed for that purpose [28]. A very useful flap to know for contractures of the first web secondary to burns and crush injuries.

A **free flap** has numerous options, and with choice, suitability increases. It's only disadvantage is that it is technically demanding and the patient has to be able to withstand considerable hemodynamic insult.

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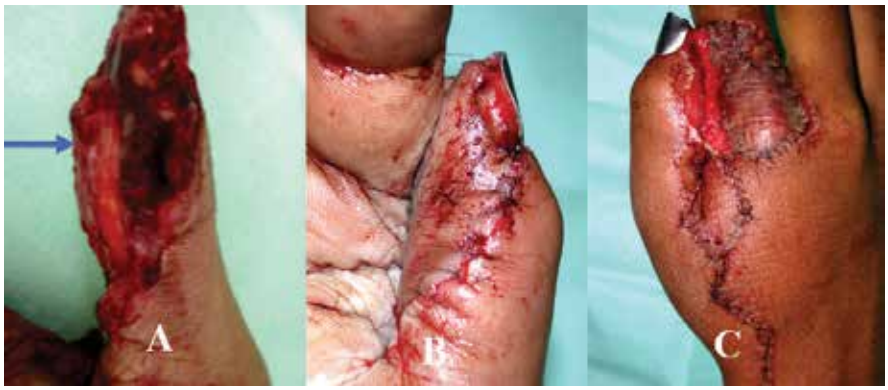

Figure 20. A: a large pulp defect of the dominant thumb in a laborer. The flexor pollicis longus (FPL) was intact as can be seen (blue arrow), but the radial neurovascular bundle was sheared off. **B:** a radially innervated cross finger (kite) flap was donated from the dorsum of the adjacent index. **C:** the subsequent donor defect was covered with a full thickness skin graft. See text for options.

Replace like skin for like skin
Mobility over dorsum of hand
Availability of tendons on dorsum
Durability (glabrous) and sensibility
Contouring of web space and fingers
Specialized structures e.g., nail complex
Preferably compatible vessel size
Avoid dependent state e.g., groin flap
Good donor morbidity

Table 9. Principles of hand resurfacing.

The principles of hand resurfacing (**Table 9**) demand that skin should be replaced by like for like, meaning glabrous palmar skin should ideally be replaced by glabrous skin. The skin on the **dorsum of the hand** should be thin and allow for the gliding of the extensor tendons. If there is a deficit of the **extensor mechanism,** then a suitable donor site for a composite flap such as the dorsalis pedis flap may be chosen. Donor morbidity is quite high though. Scheker et al. have shown, however, that tunneling extensor tendon grafts under free lateral arm or groin flaps allows functional tendon excursion [31]. These may be performed as a single stage primary procedure. Medium sized defects can be covered by medialis pedis flaps or groin flaps. The latter can cause dependent edema. Better options include the lateral arm flap, temporalis flap and anterolateral tibial flap. Extensive defects on the **dorsum of the hand or forearm** may require free flaps especially if composite tissue is required and the hand is not to be placed in a dependent position to avoid edema. These may be offered by latissimus dorsi, serratus anterior or parascapular flaps. Medium to large defects of **glabrous skin** may be obtained as a free flap from the medial aspect of the sole of the foot, at the medial arch,

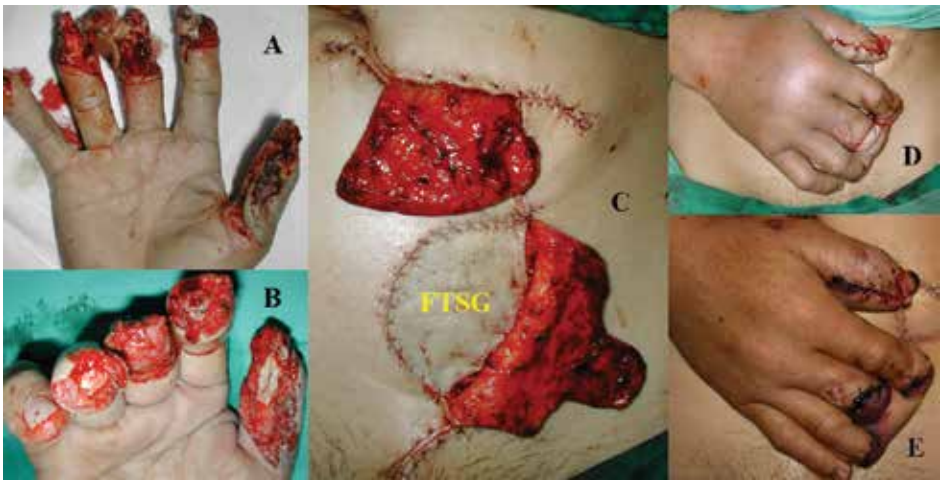

Figure 21. A: severe crushing of all digits in a 56-year-old man**. B:** postdebridement. **C:** a custom-designed abdominal flap to cover the deficit. **D:** the right hand in place. Notice the dependent edema. **E:** 20 days post op, prior to division of the flap.

in the form of either a medial plantar flap or a medialis pedis flap, while small defects on the palmar aspect may be covered by local or pedicled flaps such as a thenar, cross finger or neurovascular island flap. Replacement of the pulp may be sufficient with the former if small, but may require a toe pulp transfer if large. The posterior interosseous flap is a good flap to use, since it does not rob the forearm of one of its dual blood supplies, has an extensive coverage area and may even replace bone [32]. It is often used to widen the first web space but can be extended distally to cover just up to the metacarpophalangeal joint. However, it can be used to cover multiple digital and pulp losses by flexing the digits. Alternatively, multiple **digits** can initially be syndactylized and surfaced with a pedicled groin or abdominal flap

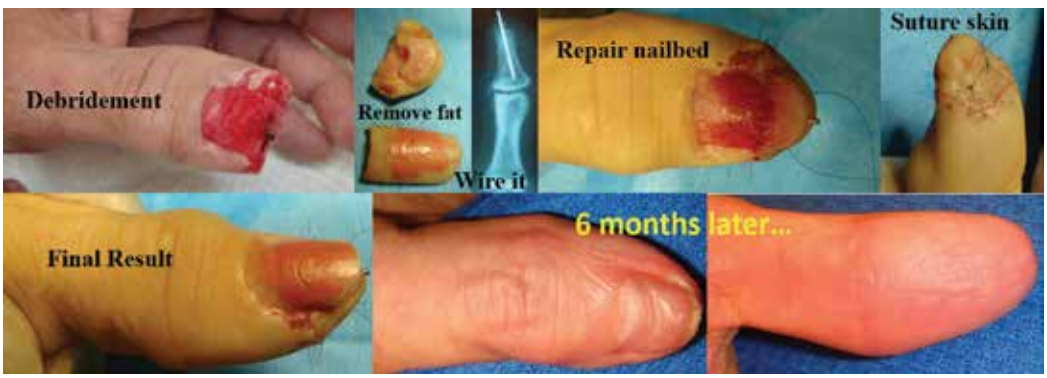

Figure 22. A 73-year-old lady caught her thumb in the door and sustained a complete amputation as shown. The amputated part was only brought in 24 h later although it was stored in a fridge. All the subcutaneous fat was removed completely and the sterile matrix and hyponychium remained. The skin edges were trimmed. The next step is to fix the remnant bone to the proximal fragment and compress it tight. The nail bed is then meticulously repaired with Vicryl 7/0. The skin is sutured with Ethilon 6/0 as a full thickness graft. Flavine emulsion dressing is applied. A reasonable good result is obtained and 2-P.D. Returns to at least 5–6 mm.

(**Figure 21**), with subsequent release. Alternative options include a medialis pedis flap, which has compatible vessel size and good donor morbidity. For single digits and smaller areas, Littler's flap, free vascular island flaps, or venous flaps may be utilized. Distally based flaps of degloved skin may be revascularized by restoring both arterial inflow and venous outflow.

The **fingertip** is composed of the nail complex and the pulp. Both of these are highly specialized tissue and require meticulously sparing debridement. The **nail complex** is composed of the nail plate, and the nail bed sitting on the distal phalanx being proximally covered by the eponychium. Nail bed lacerations should be repaired under loupes magnification with absorbable 6/0 if not 7/0 sutures (**Figure 22**), while nail bed defects may require grafting. More extensive loss may require a partial toe transfer especially in thumb reconstruction. Other options for the nail complex and distal phalanx would be to use a second toe or a great toe wrap around.

Pulp loss replacement is for cosmetic reasons similar to the nail complex. It is highly sensitive at the tip and hence prone to neuromas and hypersensitivity. Minimal loss can be replaced by V-Y, thenar, cross finger or hypothenar flaps, while more extensive loss requires toe pulp transfer [33].

4. Learning from outcomes and case examples

4.1. Fingertip injuries and amputations

These are by far the most common hand injuries seen in the emergency and may range from a simple laceration to a complete amputation of the fingertip. The anatomy of the fingertip may be accessed in many esteemed publications but the basic components that are the bone, the overlying nail bed and the volar pulp. As mentioned before, the scaffold must sustain the structure, thus it is imperative to achieve bony stability. **In children**, it may not be necessary (and even detrimental to the blood supply) to place a Kirschner wire, and up to the age of 5, the amputated part may just be capped on with gentle encircling sutures of 7/0 Vicryl or 6/0 Monosyn depending on the size of the digit (See previous chapter, **Figure 11**). The key is to reinsert the original nail plate or a synthetic material in the eponychial fold—for at least 2 weeks—to maintain it open. **In adults**, of course replantation has the best outcome [34], but if the amputation is through the midpoint of the nail plate, we find a good cosmetic outcome—with a much lower learning skill—can be achieved by performing a "cap reattachment" [35], also known as a non-microsurgical attachment, somewhat similar to a composite graft [36]. The basis we believe behind this is that all the three structures are meticulously approximated by fine sutures to provide a continuum of the blood supply, encouraging healing. First, the amputated part is thoroughly cleaned by removing **only** the subcutaneous fat from under the skin and leaving in essence a full thickness skin graft (with the edges trimmed), the bone and the hyponychium with the sterile matrix still attached to it. The bone is then K-wired together and compressed proximally. The nail bed is sutured and lastly the skin is tied over and attached as one would do a full thickness graft (**Figure 22**). A "cap" graft in our experience may be performed for crush injuries with amputations just distal to the lunula (Tamai I or Allen II) with opposition of all the three components of tissue (pulp, nail and bone) as described in [37, 38].

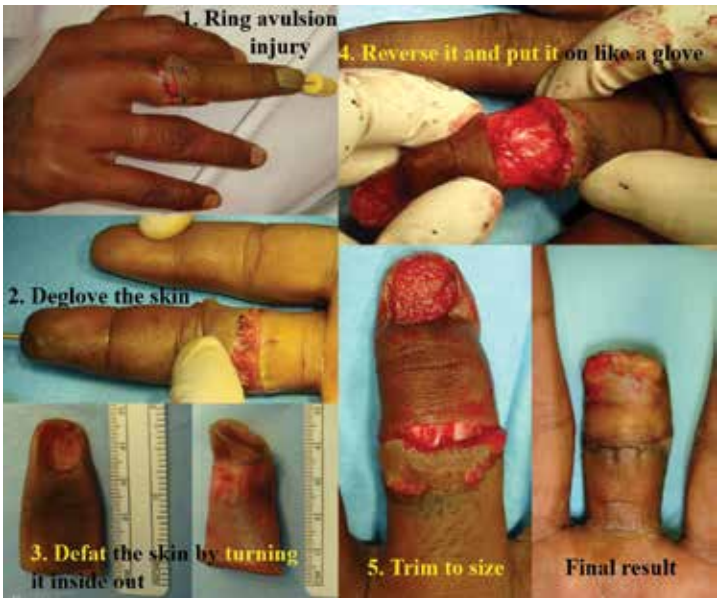

Figure 23. Ring full thickness skin graft (FTSG) in an avulsion amputation or a failed replant.

4.2. Digital amputations

Without doubt, amputations are challenging to the junior as well as the senior surgeon and the difficulty increases the more distal it is. Although replantation is the most obviously best option if it is possible and there are a number of good articles on the technique [34, 39, 40], a revision amputation may be performed with some tips and tricks in mind to maximize the function. In a ring avulsion injury where the skin and subcutaneous tissues are avulsed and the bone plus tendinous structures are intact, or not (Urbaniak II, III), a ring skin graft may be applied (**Figure 23**) and the use of some regenerating agents may prove to be helpful [41]. Wherever possible, the proximal interphalangeal (PIP) joint should be preserved, for a prosthesis is more functional at this level. Replantation of digits with amputations distal to the FDS insertion do better as opposed to zone 2 ones and Urbaniak advocates amputation [42]. In these instances, placing a full thickness skin graft in a ring fashion may be helpful because it is firmly adherent on its base, provides a good cosmetic outcome and is relatively easy to perform. Alternatively, an abdominal flap may be fashioned (**Figure 19**) or a venous flow through free flap, in increasing degrees of difficulty.

4.3. Multiple fractures and soft tissue injuries

The key is to stabilize the bony structure as sturdily and speedily as possible. Once that is achieved, the soft tissue elements will fall into place and be relatively easier to repair and reconstruct. The servile Kirschner wire may prove to be very useful as do the miniexternal fixators from LINK®. It is extremely useful to master Lister's technique as well as polishing one's skills in applying a (minilocking) plate. We have found using bone grafts (synthetic) may aid

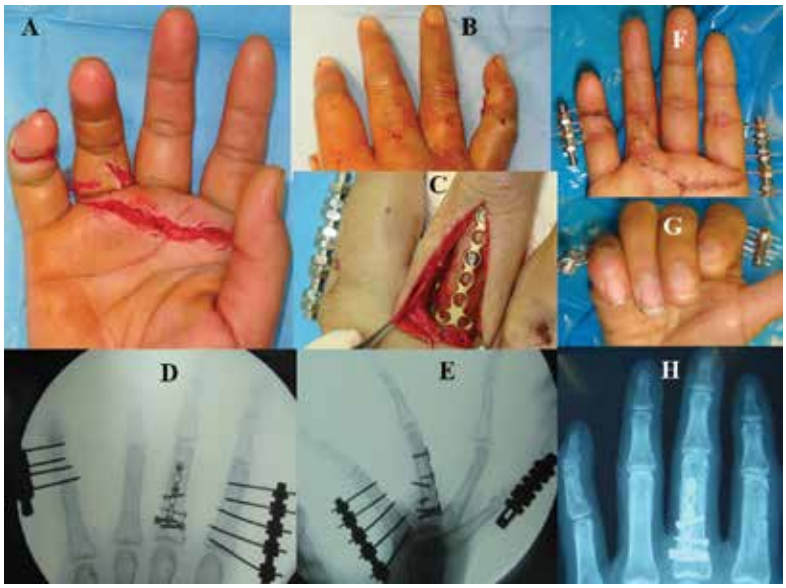

Figure 24. A, B: a crushing line of force runs across all four fingers, sparing the ring. **C-E:** miniexternal fixators were applied to the border digits—the index and small, whereas the middle was plated partly also due to extensive comminution. **F, G:** she was able to move within a week of the trauma. **H:** fixators removed after 6 weeks and plating remains.

tremendously in filling up gaps and aiding the healing process. Restoration of blood supply or minimizing use of the tourniquet is also a tip to remember. In a number of cases of forklift injury to the hand where multiple comminuted fractures occurred in one hand, we used a combination of plates, screws and external fixators to attain rapid stabilization of the skeleton (**Figure 24**). Damaged flexor and extensor tendons were also repaired in the same sitting.

5. Poor functional outcome

A poor outcome is unintentional and part of a learning curve that a surgeon goes through. There are certain pointers to avoid, which ensure known complications (**Figure 25**) do not occur. Amongst these are:

- **i.** a poor history, neglecting to ask the mechanism, the ischemic time and if the patient smokes!
- **ii.** insufficient radiographs depicting the extent of injury
- **iii.** inadequate wound debridement of devitalized and crushed tissue leading to delayed healing and infection
- **iv.** infection causing fibrosis, scarring and contracture, a sure sign of stiffness
- **v.** insufficient strength of repair of structures (such as nerve, tendon and bony stabilization) to start early active rehabilitation

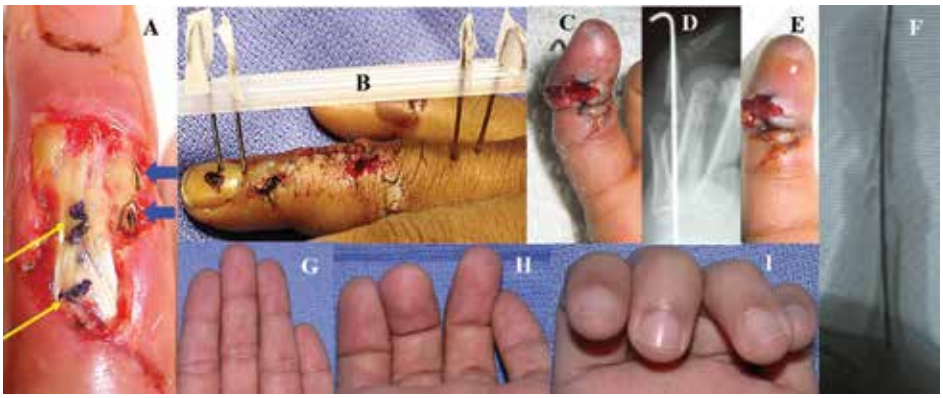

Figure 25. A: extensor tendon repair with Vicryl 3/0 (yellow arrows)—it eroded the thin overlying dorsal skin. The screw heads (blue arrows) are way too big too. **B:** K-wires passed through the germinal matrix will result in nail deformities. **C, D:** an enormous K-wire driven through a 5-year-old's little finger causing sluggish venous return. **E:** wire removal decongests. **F:** a finer wire does the trick. **G:** no malrotation. **H:** on slight flexion, the malrotation of the ring is seen. **I:** full flexion reveals more.

vi. poor vascular bed and perfusion leading to iii as well

vii. inadequate soft tissue cover especially over hardware or incorrect choice of implant size

viii. placing fixation through the germinal or sterile matrix resulting in nail deformities

ix. malunion in terms of angulation or rotation—1° of MC rotation results in 5°of fingertip malrotation [43] and 5° of MC rotation ends up with 1.5 cm digital overlap [44], thus fixation has to be very precise

x. malunion in terms of shortening—2 mm of MC shortening produces a 7° extension lag, while 1 mm of P1 shortening gives a 12°-extension lag [43]

xi. skin closure under tension will result in ischemic and necrotic tips

xii. poor hand immobilization.

Thus as can be seen, most of the adverse outcomes can be avoided. The hand surgeon has a multitude of options in resurfacing the hand. Though there are challenges to be overcome, present day microsurgical and other techniques together with biological options offer the hope of good functional outcome in crush injuries to the hand.

6. Conclusion

These are devastating injuries that are daunting; however, after the patient is stabilized and initial first aid is administered, the assessment should provide a checklist of what is damaged, and what is available for reconstruction; this would include what may be "scavenged" from the parts that are not reattachable—such as skin, parts of bone, even tendons nerves and vessels. The most challenging part is for the surgeon to accept that a "normal" hand will not result

but we must remember that if almost all is lost, whatever we salvage is a plus point. Having said that, there are some main points to bear in mind—the goal should be the "greatest total benefit for our patient" [45], which may be different in various countries, cultures and beliefs. Clear communication, an idea of what is feasibly possible and the likely end result are some of the factors to bear in mind when discussing options with the patient and their relatives.

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Conflict of interest

The authors declare no conflict of interest and have not received any remuneration or benefit from any entity for the writing or publication of this article.

Appendix & nomenclature

2-PD:	two-point discriminator
ADM:	abductor digiti minimi
DIPJ:	distal interphalangeal joint
FDP:	flexor digitorum profundus
FDS:	flexor digitorum superficialis
K-wire:	Kirschner wire
MCPJ:	metacarpophalangeal joint
MESS:	Mangled Extremity Scoring System
MVA:	motor vehicle accident
P:	phalanx 1: proximal, 2: middle, 3: distal
PIPJ:	proximal interphalangeal joint
PT:	post-trauma
ROM:	range of motion
RTA:	road traffic accident

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